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MODERN TRENDS
IN
ACCIDENT SURGERY
AND MEDICINE

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and

TO THE MEMORY OF OUR FRIEND AND COLLEAGUE
Ruscoe Clarke

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PREFACE

TENDING the injured was probably the origin of medicine and was certainly the beginning of surgery and even in its ancient beginnings surgery was concerned with the medical state of the injured patient as well as the treatment of his wounds. The unknown Egyptian authors of the Edwin Smith papyrus described the differing effects of injuries on different parts of the body and classified them into those which should be treated and those in which prognosis was so bad that responsibility for treatment should not be accepted. Paré, who had a wide understanding of the clinical features of open wounds, also gave an excellent description of traumatic shock.

The contents of this book could not be restricted to accident surgery in the narrow sense of the term its title *Modern Trends in Accident Surgery* and *Medicine* is meant to imply that a modern understanding of accident cases involves the patient as a whole, that is the medical physiological pathological as well as the surgical aspects. Consideration and treatment must extend beyond the injured part to the general upsets in the circulation and in metabolism the origin of medical complications (renal, infective embolic and others) must be understood, treated if possible or better prevented.

Like the other volumes in the *Modern Trends* series this book does not pretend to be comprehensive. Our difficulty has been what to leave out and not what to include. Burns¹ and anaesthesia² have not been included as they have been specially considered elsewhere by members of the staff of the Birmingham Accident Hospital. The topics dealt with reflect not only their special importance but also much of the individual and collective experience and interests of the staff of the Birmingham Accident Hospital. We leave it to the reader to decide whether our judgment has been sound. All the contributors are or have been closely associated with the work of the Birmingham Accident Hospital and therefore this book also reflects much of the current practice of the hospital and its teaching through the Institute of Accident Surgery. We are indebted to our colleagues for their co-operation and help in making this possible.

Nearly all the illustrations are original and are related to case material of the hospital. They were prepared in the Photographic Department of the hospital and we are indebted to Mr R. Gill and his colleagues for their industry and skill. It is a pleasure to acknowledge the help of our medical secretaries for the tireless job of producing accurate copies of our manuscripts and to thank our publishers particularly Mr J. K. Burgess and Mr L. E. Rayner of the Medical Publishing Department, for their help and courtesy.

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July 1 1959

The sudden and untimely death of Ruscoe Clarke occurred soon after this book was completed and while it was in the press. All who worked with him will remember with admiration his untiring work and devotion to accident surgery.

August 1 1959

¹ Sevel, S (1957)
² Walton, L. J.

Burns: Pathology and Therapeutic Applications. London: Butterworths.
Anaesthesia for the Injured. In the press.

CHAPTER 1

ORGANIZATION OF ACCIDENT SERVICES

THE EDITORS

AN IMPORTANT medical challenge of our time is the necessity to abolish architecturally archaic casualty departments and associated old fashioned notions these must be replaced by an efficient system of accident services based on modern ideas of need and design Too long has the treatment of accidents been the Cinderella of our hospital system and although the last 50 years have seen remarkable advances in medicine and surgery the treatment and study of accidents has lagged behind An interest in wounds, haemorrhage and casualties has periodically revived in time of war but has tended to flag when hostilities ceased Yet gradually the appreciation has grown that it is necessary to apply modern techniques and advances to the treatment of civilian injuries. This is not easy to implement without solving a number of problems of organization

An efficient organization must be based on two interrelated principles (1) the formation of accident departments in hospitals through the segregation of accident cases and (2) the availability of a whole range of medical surgical pathological and radiological services almost at a moment's notice.

The slowness in developing proper accident services was originally due to a resistance to the separation of injured patients from the stream of general surgical services later when the concept of fracture clinics was accepted, it resulted from the notion that it was impossible to unite the treatment of fractures with the treatment of injuries to the cranial thoracic or abdominal contents Yet trauma does not recognize neat anatomical or regional divisions and injuries are often multiple, requiring simultaneous treatment consider for example, the frequency of limb fractures in patients with head chest, spinal or abdominal injuries and the frequent combinations of head and chest, chest and spine or chest and abdominal injuries. The patient must be considered as a whole and, though often treated by a team, must come under the over-all care of one surgeon. Delay in development also arose from difficulties financial and otherwise in planning for treatment within a short time of injury and, before 1948 from the lack of an organized and integrated health service The present national system in Great Britain of Regional Hospital Boards provides the opportunity for full development of highly efficient accident services worthy of the country's medical tradition provided that the will is present.

MAGNITUDE OF THE PROBLEM

The modern accident problem stems largely from social and industrial developments of the nineteenth and twentieth centuries A considerable number of accidents arise from industrialization and the rapid increase in motor transport, whilst increased longevity has added to the incidence of certain injuries in the aged

Among a population of about 50 million people in Britain about 20 million injuries are treated every year outside hospital by medical, nursing and first aid services. These are of course, mainly minor injuries. In addition about 2 million casualties are treated in hospitals (Gissane 1955).

In 1958 road casualties totalled 293 797 persons injured and 5,970 persons killed. This included 49 146 children (under 15 years) injured and 717 killed and 31 631 elderly people (over 60 years) injured and 1 768 killed. The total injured was the highest on record and the total of deaths the largest since 1944. The children's figures were higher than at any time since 1949 when they first became available.

In 1957 reportable factory accidents (excluding the docks and mines) numbered 174 713 with 651 deaths, even though there was a tendency for industrial injuries to fall in the previous decade.

Figures relating to domestic and other accidents are difficult to obtain. The Registrar-General's Statistical Review for 1956 attributed 3 498 deaths to accidents by falling at home of these 3 173 were in elderly persons (over 65 years).

Children

The most serious injuries in the younger age groups result from road accidents and falls from heights. Children also sustain a high proportion of the more dangerous burns.

It is important to recognize that injury is the largest single cause of death in every age group from 1 to 39 years. For example the death rate from accidents in boys 5-14 years of age is higher than the combined rates from tuberculosis, pneumonia, heart disease, rheumatic fever and other infectious diseases.

Fatal Injuries

A survey of the deaths in the Birmingham Accident Hospital showed that most of the injuries dangerous to life can be divided into four main groups (Clarke 1959).

Head injuries

Three-quarters of the deaths from head injuries occur among males. Many of these have injuries elsewhere and sometimes the associated injury is sufficient to be a potential cause of death. Many cases are fatal within a day or two of accident but many others succumb to complications which are potentially treatable or preventable.

Fractures of the femur in the elderly

The second group comprises deaths following fractures of the femur in elderly people, mostly females. Most of these deaths are from supervening bronchopneumonia or pulmonary embolism. The latter is largely preventable by prophylactic anticoagulant therapy (see Chapter 17).

Miscellaneous

A large miscellaneous group includes many younger people with abdomino-thoracic injuries or severe internal bleeding. Many of these injuries are not

necessarily fatal provided that first-class medical and surgical attention is rapidly available.

Burns

Burns are a special problem (see Scvitt, 1957)

FRACTURE CLINICS OR ACCIDENT CENTRES

The recent history of this controversy began in 1913 with a report on the treatment of fractures by the British Medical Association. Soon after this a number of orthopaedic centres and a few fracture clinics in general hospitals were developed, partly stimulated by World War I and the efforts of Robert Jones and his associates. The foundation of the Unfallkrankenhaus in Vienna in 1926 under the direction of Dr L. Böhler was a great stimulus because for the first time a major civilian hospital in a large city assumed responsibility for treating injuries and only injuries. Its economic support was derived from insurance companies: this pattern of finance never developed in Britain but took root sporadically elsewhere in Europe and in America. The need for organized fracture services was again stressed by another report from the British Medical Association (Report, 1935) and in a report by a government Interdepartmental Committee (Report, 1939) which stated: "surgical opinion is agreed in recommending the institution of special fracture services. The system which was universal to a recent date by which fractures are treated in general surgical wards under general surgical routine is gravely defective." Recognition was given to the importance of after-care and full rehabilitation, to the need for a special organization within a general hospital to cover treatment from the time of injury to restoration of full working capacity and to the necessity to continue in-patient and out-patient treatment in a single department by the same team. The idea that a full traumatic service was impossible without a team of surgical "super specialists" was expressed to the committee by the late Sir Cuthbert Wallace who said: "Traumatic surgery covers such a lot of ground. A man may have a crack on the head or a ruptured intestine. You cannot expect a man to specialize in all these things. They belong to different departments. By talking of traumatic surgery you are mixing up a lot of things." Nevertheless, the committee did not oppose the development of full accident services but stated that "the most important and most urgent need at present is the provision of services for the treatment of fractures and our recommendations bear on the steps necessary to secure this. Advance towards the wider conception of a traumatic unit (if found desirable) is more likely to take place by a process of development than by pressing the conception at once."

A fuller accident service was recommended by the British Orthopaedic Association in 1943 in its Memorandum on Accident Services.¹ This emphasized that "the casualty departments of general hospitals must be replanned, reorganized and often rebuilt. There must be a separate organization and even a separate door of entry for accident cases on the one hand and acute medical and general surgical emergencies on the other. In the accident department many cubicles should be available where patients are prepared, examined and treated for shock. There must be immediate access to radiographic departments leading directly to emergency theatres fully equipped for the excision of wounds and the emergency treatment of injuries."

¹ For its new memorandum, see Addendum on page 8

ORGANIZATION OF ACCIDENT SERVICES

With all this we agree and to it would add provision of a 24-hour laboratory service particularly for the technical aspects of blood transfusion. The memorandum rightly drew attention to the necessity of hospital beds for "minor" injuries, the need for follow up clinics and continuity of treatment, and to other matters of organization but it had certain shortcomings. Trauma was considered fundamentally a part of orthopaedic surgery the development of accident services was seen as an extension of fracture departments although the treatment of visceral injuries was not excluded. Accident departments in general hospitals were envisaged rather than independent accident units. The report in fact outlined the development which has slowly taken place since the inception of the National Health Service in 1948.

BIRMINGHAM ACCIDENT HOSPITAL

One important exception was the foundation in 1941 of the Birmingham Accident Hospital under the direction of Mr W Gissane. It took over an old hospital building in the heart of the city and, starting with limited staff it treated 10 000 patients in its first year later it received wounded servicemen from the Second Front of World War II. It now receives 50,000 new patients every year about 7 000 are in patients and of these about 5 000 are admitted to short-stay wards. Part of the old interior was redesigned and provided a new type of casualty and admission department with a planned layout to facilitate the out patient care of wounds and fractures. The casualty department and the in patient work are integrated and not separated by artificial barriers of organization. A simple but new kind of dressing station for the out patient care of minor cuts and lacerations was provided in order to eliminate or greatly reduce the incidence of added bacterial infection this was based on a no-touch technique and the physical separation of "clean" and "dirty" wounds, staff instruments and dressings. The hospital includes a self-contained burns unit with its own staff closely associated with a Medical Research Council unit, a radiological service and a large rehabilitation department providing physiotherapy remedial exercises and occupational therapy.

Treatment is comprehensive and continuous and is carried through to the fullest functional recovery. Surgery includes acute and reconstructive orthopaedic, abdominal thoracic, intracranial, plastic, peripheral nerve and vascular operations. Pathological services have grown with the years and cover a comprehensive range of the various disciplines now found so necessary in treatment, prophylaxis and research. A day and night emergency laboratory service, particularly for blood transfusion, is, for example just as necessary as radiology and operating theatres. Biochemistry is needed for the understanding and treatment of electrolyte metabolic, renal hepatic and other disturbances. Bacteriology is essential for the prevention and intelligent treatment of wound and other infections. Laboratory control of therapy is needed for prophylaxis and treatment of thrombo-embolism, whilst histology sheds light on ununited and pathological fractures, false neuromas, and disorders of joints, bone muscle and other tissues. It is also necessary to mention that patients with diabetes mellitus, pernicious anaemia, carcinoma and other diseases for whom laboratory investigations are needed are just as likely to be injured as others.

DESIGNATED HOSPITALS

Surgical organization is based on whole-time surgeons who form three parallel teams and, in addition, a team staffing the burns unit. Each team is responsible for new patients attending hospital within successive 24-hour periods. Each team has its own medical secretaries and an almoner who is responsible for organizing a wide variety of social help for the patients including resettlement, if necessary through the Ministry of Labour, the Disablement Resettlement Officers and Industrial Rehabilitation Units.

The organization allows experienced surgeons to take personal responsibility for treating major injuries from the resuscitation phase onwards and gives all the surgical staff a wide experience of different injuries.

In our experience the early treatment of many injuries, particularly road accidents, involves much more than a knowledge of orthopaedic or plastic surgery; resuscitation requires surgical judgment which cannot be delegated to anaesthetists or transfusion officers except as part of a team. The surgeons must develop a general knowledge of the effects of injury in all parts of the body and be able to assess priorities; patients with multiple injuries cannot be sorted early and handed over to appropriate specialists. The initial treatment requires surgeons with a wide knowledge of many fields and special experience in injuries. Each consultant surgeon must be experienced in the orthopaedic care of fractures, the treatment of wounds and abdominal surgery and have a sound working knowledge of the traumatic aspects of cranial, thoracic and plastic surgery. It is irrelevant whether he is called a traumatic surgeon, a general surgeon with traumatic experience or an orthopaedic surgeon with wider interests. It is the man, his interests, training and experience and not the terminology that matters. This does not mean that there is no place for the specialist thoracic or plastic surgeon or neurosurgeon in traumatic work. On the contrary they often play a vital part; their advice and skill must be available readily and fully used when required.

The segregation of accident cases has facilitated advances in treatment, and the experience of the Birmingham Accident Hospital has sharply focused attention on many problems. It is now clear that the organization needed for treating injuries on a regional or national basis requires more than the extension of fracture services. Nevertheless the relative isolation of the hospital from a medical centre has brought certain disadvantages. Contact with other specialities and disciplines is not always easy and certain problems of staffing would be solved more readily in an autonomous or semi-autonomous unit that was part of a wider hospital organization.

DESIGNATED HOSPITALS

The multiplicity of hospitals in cities and the unevenness of standards in the treatment of injuries has focused attention on the necessity to designate a limited number of them as special accident units or centres; these alone should receive casualties brought in by ambulance. In considering the London accident services the *Medical World* (1956) wrote: "Instead of an accident service we have a complicated jig-saw of more or less integrated sections—hospitals of every size, type and quality; doctors with every variety of specialized training or lack of it; responsibility divided between half a dozen public authorities. The answer is obviously to reduce the number of casualty-receiving hospitals and to concentrate the casualty resources into these." Segregation of accidents in one or more designated

ORGANIZATION OF ACCIDENT SERVICES

hospitals providing treatment as well as sorting, is essential for organizing an efficient round-the-clock emergency service in an urban area. Experienced medical staff must be available at short notice. Facilities for safe blood transfusion and for radiology must be on a 24-hour basis and operating theatres must be available without delay. Ambulances would be instructed to take accident cases, not to the nearest hospital but to the nearest designated hospital that is, one provided with an accident department. This principle received wide support at the combined meeting of the sections of Traumatic Surgery, Orthopaedics and General Surgery at the 1958 Annual Meeting of the British Medical Association.

NATIONAL AND REGIONAL ORGANIZATION FOR ACCIDENTS

A nationwide accident service must have a regional basis. We envisage (1) at the centre of each region an accident hospital or a large accident block of a big general hospital, (2) a peripheral service based on accident departments in designated general hospitals, and (3) further to the periphery the full up-graded services of general practitioners. The hospital services should work closely with the casualty services in industry. We consider it essential that treatment as it is under the National Health Service in Britain, should be "free" in the sense that it is provided for by general taxation rather than payment at the time of need.

This plan has repercussions on the structure of medical practice. Full facilities for treating minor injuries by general practitioners would not only relieve the hospitals of a large burden of work but would prevent many minor injuries from developing major complications. This can be properly implemented only through group practice, preferably in health centres which could provide facilities for minor surgery and radiology. In rural areas some kind of group practice would permit a practitioner to attend to acute emergencies reaching the cottage hospital without detriment to his other work. At the factory level an extension of efficient casualty departments in large and medium-sized factories is needed whilst the possibility of sharing a casualty department amongst a group of small factories should be further explored. This involves the problem of an industrial health service and its links with the National Health Service.

General hospitals properly staffed and designated with accident departments would treat most of the casualties needing hospital treatment.

The central accident service in an urban area could be provided either as an independent accident hospital on the lines of the Birmingham Accident Hospital or alternatively as a semi-autonomous large wing or block attached to a major general hospital, preferably an undergraduate teaching hospital. Such a link with the medical school would stimulate a more comprehensive teaching of undergraduates in the treatment of accidents and so equip the general practitioners and surgeons of the future. The advantages of an accident block over an accident hospital are first, the ready availability of a variety of medical, surgical and other specialists in the other parts of the hospital; secondly, the possibility of rotating nurses, radiographers, medical and other staff and so solve problems of staffing; thirdly, the possibility of training larger numbers of personnel. The accident block must, of course, have the continuous services of consultants, otherwise there is a danger that it might become a backwater with junior staff working in isolation and

MASS CASUALTY PROBLEM

losing their enthusiasm A third possibility is an emergency hospital such as the Moscow Emergency Hospital which receives all kinds of medical and surgical emergencies including the injured. Such a scheme could upgrade the treatment of all emergencies although the multiplicity of disciplines and skills required for first class treatment might make it impracticable except in very large cities. It involves complete central control of all ambulance services and emergency admissions to other hospitals.

The functions of a central accident hospital or block would be as follows

- (1) To provide a full accident service on a neighbourhood or area basis
- (2) To provide a consultant accident centre for the accident departments of the designated hospitals. It would receive special cases from them on a regional basis and advise on various matters in emergency by telephone or by radio to ambulances or even to the scene of an accident
- (3) To admit and treat the difficult cases of burns and scalds in the region through the inclusion of a properly designed burns unit.
- (4) The training and teaching of various kinds of staff through secondment or rotation, thereby upgrading the regional service as a whole
- (5) Research into new methods of diagnosis and treatment including not only short term projects but also long term and fundamental research problems.
- (6) General postgraduate teaching of surgeons-in training, general practitioners, industrial medical officers, nurses and those responsible for teaching first aid.

These special duties and functions mean that the centre must be medically overstaffed compared to its complement of patients or beds

AMBULANCE AND FIRST AID SERVICES

Our present services are undoubtedly efficient in that little time is lost in transporting a casualty to hospital but the official links between the ambulance and first aid services and the hospitals are few. All could benefit by a closer liaison and the teaching of first aid could be improved. The provision of accident services to isolated rural communities is a special and difficult problem. It cannot be considered here except to say that full use should be made of modern helicopters either transporting the patient to hospital or sometimes a team of doctors and their equipment to the casualties.

MASS CASUALTY PROBLEM

Civilian accident services must be concerned with the problem of mass casualties from disasters like train crashes and aeroplane crashes in populous areas and some attention has already been paid to this. The basic need is for rapid evacuation of seriously injured patients to hospitals where full treatment is available. Designated hospitals and the special accident centres should be readily adapted for emergency expansion. If possible one hospital should receive and sort the first 50-100 patients whilst other hospitals are being mobilized should additional help be needed. The more experienced surgeons will be best occupied at hospital level. When victims are trapped or cannot be moved, surgical units may have to be sent to the scene of the disaster. A mobile transfusion unit and facilities for major first aid should be available. Nevertheless, seriously injured patients should rarely be held at the

scene of the accident they should be immediately transported to an appropriate hospital if this is situated within 30-40 minutes driving distance provided treatment is begun immediately after arrival (Clarke 1955). Otherwise resuscitation before and during transport may be needed and group O blood should be transfused (see Chapter 6).

Addendum

The new memorandum on accident services by the British Orthopaedic Association (1959) appeared when this chapter was in the press. It advocates that the organization of accident services must be undertaken by the state as a quasi-military operation and that considerable expenditure is needed over many years. A nation-wide, 24 hours a day accident service is proposed. In each area there would be a central accident unit attached to a general hospital and this, together with the upgraded services of peripheral hospitals would provide the accident service for the area. The accident unit would be fully staffed and have facilities for the reception, resuscitation and continued treatment of all injured patients. It is estimated that the accident units would need 150-250 beds for half a million of population assuming that beds for convalescence, especially for the elderly are also available. At the peripheral hospitals there must be a clinician, either a resident surgeon in a small town or a general practitioner who can deal competently with minor injuries and who can distinguish between these and injuries which are actually or potentially serious. In principle these proposals are similar to ours. However accident surgery was again envisaged as an extension of orthopaedic surgery and although team-work was stressed, "the orthopaedic surgeon should confine his responsibility to injuries of the limb and spine the neurosurgeon will deal with injuries to the head, the thoracic surgeon with injuries to the chest, the abdominal surgeon with visceral injuries and so on". They still consider that it is not feasible and even undesirable to have surgeons who accept responsibility for all the injuries of the patient even though this is a basis of the surgical success of the Birmingham Accident Hospital.

REFERENCES

- British Orthopaedic Association (1943) *Memorandum on Accident Services*
 — (1959). *Ibid.*
 Clarke, R. (1955). *Med World Lond.*, 82, 282.
 — (1959) *Brit med J.*, 125
 Gissane, W. (1955) *Practitioner* 175 450
 Medical World (1956) Editorial 127
 Report (1939) *Rehabilitation of Persons Injured by Accidents. Final report of the Interdepartmental Committee* London H.M. Stationery Office.
 Report of Committee on Fractures (1935) *Brit med J* Suppl 1 53
 Sevitt, S. (1957). *Burns, Pathology and Therapeutic Applications* London Butterworth.

CHAPTER 2

SYSTEMIC REACTIONS TO INJURY

S SEVITT

THE PURPOSE of this chapter is to put into perspective a number of important general effects of injury some of which are discussed more fully in other chapters. All severe injuries are followed by a series of complex and often interacting changes which affect tissues and organs remote from the site of insult. The problem of thresholds of severity below which such reactions do not occur is vexed and difficult.

Many of the reactions are parts of an integrated series affecting the nervous cardiovascular endocrine and other body systems. Sometimes the various causes and effects are difficult to unravel since one body system may be affected by or produce changes in the others. The acute oligæmia following hæmorrhage and severe injury is not only responsible for post traumatic anaemia and much of the circulatory syndrome known as shock, with its dangers to the general renal and cerebral circulations, but is, for example, a sustaining cause of the adrenocortical secretion of aldosterone and probably other steroid hormones. The body reactions triggered off by injury are determined by the severity nature location and circumstances of the trauma and are influenced by therapy. Adequate replacement of blood loss not only restores the general circulation and prevents anaemia but also reduces the abnormality in the excretion of electrolytes and water (Fleair and Clarke 1955) and certain disturbances of iron and hæmoglobin metabolism (Baar and Topley 1956).

Teleologically the reactions to injury may be considered either as homeostatic or defence mechanisms or serving both purposes. Regional, particularly renal and splanchnic, vasoconstriction maintains the systemic blood pressure and the cerebral and coronary circulations for a time against a falling cardiac output. hæmodilution and post hæmorrhagic sodium and water retention by the kidneys combat the loss of blood volume. the constriction of skin and muscle vessels reduces bleeding and conserves body heat. Reactions such as these must have been developed through the ages to maintain survival of the species but their benefit is limited and beyond a certain point some give rise to pathological disorder. The limitation of the body's homeostatic and defence mechanisms is the price paid by individuals for group survival.

The various body reactions must be considered separately but integration of function and interrelation of activity must always be borne in mind.

NERVOUS ACTIVITY

Acute nervous irritation reflexly induced from the injured area was at one time considered the cause of traumatic shock but although there are still protagonists of this view it is now generally agreed that oligæmia is the most important single

factor. This does not mean that nervous dysfunction is unimportant. Emotions like fear or excitement and pain may produce primary or neurogenic "shock" such as a collapse or faint which is normally rapid in onset and recovery. Nervous activity initiates this reaction during which pallor, sweating, vomiting, hypotension and bradycardia are common features. Syncope is probably the result of a redistribution rather than a fall in the cardiac output (McMichael, 1944).

The relationship of primary neurogenic symptoms to those of oligæmia varies in different patients. Sometimes recovery from primary "shock" is complete and there is an interval before the signs of oligæmia appear. Often primary neurogenic effects are trifling and the first major symptoms are those of oligæmia, whilst in other patients neurogenic "shock" may be superimposed on the effects of oligæmia thereby confusing diagnosis. When blood loss is rapid it may be impossible to separate primary neurogenic and oligæmic effects. Much of the cardiovascular response to oligæmia is mediated by nervous reflexes arising within the vessels and acting on the heart and vascular bed. The resultant clinical picture may be similar to that of a primary neurogenic reaction and no doubt this has been a cause of confusion.

Sympathetic overactivity is generally a prominent feature of oligæmic shock and is manifested by skin pallor, tachycardia, regional vasoconstriction and possibly sweating and the absence of cutaneous vasomotor reflexes. Sympathetic stimulation perhaps with coincident parasympathetic depression accounts also for decreased salivation, fall in gastric acid peptic secretion and the decreased stomach motility which occur after major injury. A reduction in the stomach-emptying rate after a meal was found in rats subjected to muscle (tourniquet) ischaemia (Whiteley and Green, 1952). Lethal ischaemia suppressed stomach activity whilst sublethal ischaemia delayed the emptying for a few hours. The reduced motility of the stomach is important to anaesthetists who may have to deal with a full stomach even as late as 24 hours after a meal. Autopsy of patients dying after major injury may show gaseous distension of the proximal colon and often the stomach, corresponding anatomically to the distribution of the sympathetic nerves and consistent with sympathetic overactivity. Sheehan (1948) reported similar findings in obstetric shock. There may be an increased excitability of cardiodepressive reflexes which could account for the cases of cardiac depression during tracheal intubation for anaesthesia. The brain becomes more sensitive to anaesthetic agents and much smaller doses will suffice in shocked patients when inadequately transfused (Wolfson, 1957). The results of additional nervous activity may complicate or modify the common reactions to oligæmia. These include phases of hypertension which are not uncommon, an unsustained period of hypotension, the occasional development of bradycardia and variations from case to case in skin pallor, sweating and vomiting. Higher nervous activity from apprehension, pain or excitement are no doubt important but cannot be the whole explanation since acute hypertension for example, may occur in sleeping or co-operative patients. Such hypertension is often abolished by anaesthesia, probably through vasodilatation in muscles and by ganglion blocking agents such as hexamethonium compounds. Reflex stimulation from the injured area probably occurs; this is the basis of the treatment of war wounds with local anaesthetics advocated by Vishnevsky (1941). He thought that shock was largely an acute metabolic

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derangement set in motion by local nerve impulses and that the tissue "dystrophy" could be corrected by regional nerve block with procaine

Central and reflexly initiated nervous activity are also involved in the response of certain endocrine glands, particularly both parts of the adrenal gland and possibly of other organs, such as the kidney. Rapid secretion of adrenaline and sympathetic overactivity is the basis of the primitive emergency reaction described by Cannon (1915) and secretion of the various hormones of the adrenal cortex plays an important role in certain homeostatic and defence mechanisms (see page 18)

CARDIOVASCULAR FEATURES OF TRAUMATIC SHOCK

(See also Chapter 4)

The term traumatic shock still usefully describes the complex cardiovascular changes which may follow injury and which are mainly related to an acute oligæmia (hence the term "oligæmic shock") from overt or latent hæmorrhage. Similar clinical syndromes may occur in other conditions such as coronary thrombosis and pneumonia.

The hæmodynamic effects of trauma were studied during World War I by Bayliss and his colleagues (1919) during World War II by Cournand and his colleagues (1943) Grant and Reeve (1951) Lauson, Bradley and Cournand (1944) Noble and Gregersen (1946) and by Richards (1943). The essential features are oligæmia, a fall in cardiac output and regional vasoconstriction. A certain degree of oligæmia can be tolerated without reduction in the cardiac output; beyond this the output falls and produces a diminished flow of blood through the body and consequently a reduction in the carriage of arterial oxygen with its attendant risks of anoxia. The reduction in cardiac output is not necessarily proportional to the degree of oligæmia and the cause of this requires further investigation, for example myocardial function, ionic effects, or nervous activity may be involved. At first the arterial pressure remains normal or may even be raised owing to regional vasoconstriction; this increases the overall peripheral resistance to the flow of blood, reduces the vascular bed and maintains the circulation through the un-constricted cerebral and coronary vessels. Vasoconstriction affects the very vascular renal and splanchnic liver beds as well as the skin and muscles; if it did not occur the circulation through all organs including the brain and myocardium, would fall in proportion to the fall in cardiac output.

Vasoconstriction can only compensate up to a point for the fall in output; after this the arterial pressure falls and then the cerebral and coronary circulations are reduced. Cerebral ischaemia and anoxia is the cause of the mental confusion or semi-coma in many shocked patients. The beneficial response to transfusion is of great help in distinguishing these symptoms from those of head injury or the fulminating form of cerebral fat embolism (see Chapter 14).

Significant hypotension (systolic pressure at 100 mm. of mercury or less) within an hour or two of injury often means that a blood transfusion of 25-30 per cent of the blood volume is required to correct oligæmia. The arterial pressure may fall rapidly or after an interval; the hypotension may stabilize at different levels but the equilibrium is unsteady because a further reduction in blood volume may occur or because the reduced cerebral and coronary circulation may impair cardiac

efficiency. A sustained fall of systolic pressure to 70 mm. of mercury or less usually indicates that a transfusion of 50 per cent or more of the blood volume is needed. Sustained hypotension is a relatively late sign of shock and generally means that the circulation is in danger. Early and adequate transfusion is essential if the dangers of prolonged ischaemia of the kidneys and other organs are to be reduced.

Vasomotor decompensation

Vasomotor adjustments in acutely oligaeamic patients are strained and unstable and are likely to be upset by an unfavourable environment or wrong treatment. Sudden circulatory failure may be precipitated by alcohol, excessive heating, exposure to cold, operation and anaesthesia without transfusion, pain, movement and even changes in posture. In patients with latent or threatened shock the blood pressure may fall and the full clinical signs and symptoms of shock develop rapidly whilst patients already clinically shocked may seriously deteriorate.

The mechanisms of decompensation are imperfectly known but in some cases regional vasodilatation from the giving of alcohol, warming of the skin or general anaesthesia is probably important. This draws blood away from the heart and brain, lowers the peripheral resistance and reduces the arterial pressure.

Irreversible shock

The majority of patients improve with blood transfusion but occasionally the circulatory changes persist or worsen and are apparently irreversible by transfusion or other means. The condition must be distinguished from the inadequate effects of under transfusion with which it was confused before the large volumes of blood needed for badly injured patients was appreciated. Irreversibility cannot be diagnosed until the patient fails to respond to truly adequate volumes of transfused blood. One or more of the following causes may be responsible.

Prolonged shock—The duration of shock has been prolonged and irreversible cerebral or myocardial changes, or both, have occurred. Autopsy examination of patients dying after severe injury often shows a streak of subendocardial haemorrhage on the left side of the interventricular septum and about an inch below the aorta near the termination of the left bundle of His (Fig. 1). A similar change was reported by Sheehan (1948) in obstetric shock. This may have relevance to a complicating cardiac dysfunction.

Complications—An unsuspected injury or complication is present. The latter include (1) air embolism in cases of chest injury owing to penetration of the lung by a fractured rib and the sucking of air into pulmonary veins; (2) pneumothorax; (3) bilateral adrenal apoplexy which may occur after thoraco-abdominal compression injuries (Sevitt, 1955a); (4) fulminating cerebral fat embolism (see also Chapter 14); (5) sympathetic paralysis due to spinal injury and a little later (6) fulminating infections such as anaerobic myositis and peritonitis.

Citrate intoxication—The large amount of citrate in a transfusion of stored blood given rapidly and in large volumes might precipitate acute cardiac failure by reducing the ionized calcium concentration of the plasma (see also Chapter 4). The possibility is increased in hepatic disease because citrate is normally metabolized by the liver.

Coronary occlusion—Coronary occlusion through thrombosis or spasm is occasionally associated with the injury but it is often uncertain whether it precedes

FIG 1—Left ventricle opened showing subendocardial haemorrhage (dark area) on the left side of the interventricular septum, the "shock" lesion. This patient died 2½ hours after severe injuries to the head and multiple fractures.



or follows trauma. The risk may be increased in injured patients with coronary atheroma because there is experimental evidence that the plasma develops vasoconstrictor properties after an acute haemorrhage and coronary vasospasm might be related to this.

Shock promoting substances—The release of shock promoting substances such as adenosine triphosphate (ATP) and a vasodepressive substance (VDM ferritin) or bacterial toxins (see Fine 1952) from ischaemic liver may modify the circulatory responses. ATP is the shock producing factor in muscle extracts (Green, 1943). Significant increase in the blood of its various components (adenosine phosphate and pentose) occurs after major injury which indicates release from muscle or other cellular stores (Green and Stoner 1950). Nevertheless ATP is unlikely to exist as such in the blood because it is rapidly decomposed by dephosphorylase and other enzymes. VDM is said to be released by ischaemic liver it acts on arterioles and counteracts the excitatory effect of vaso-excitatory substance (VEM) from the kidney (Shorr Zweifach and Furchgott, 1948 1951). It has been suggested that excess of VDM may precipitate peripheral circulatory failure. Assay of VDM and VEM in the plasma of battle casualties often revealed high titres of VDM but no correlation was found with refractory shock, clinical state or prognosis (Scott, Olney and Howard, 1955). Failure to respond to transfusion was not related to the titre found.

CHANGES IN THE BLOOD

Red cell loss

Blood loss after injury has been studied by Crosby and Howard (1954) Dacie and Homer (1946) Grant and Reeve (1951) Topley and Clarke (1956), Vaughan (1948) the author and others. There is an acute loss of red cell mass which at first can only be detected either by measurements involving the labelling of red cells with ^{32}P or ^{51}Cr or indirectly from the blood haematocrit after measuring the

plasma volume by the Evans blue technique. The haemoglobin value and red cell count of the peripheral blood fall when the blood is diluted with interstitial body fluid but haemodilution may not be recognizable for a period of hours. It is established by 24 hours after injury but is not complete until 3-6 days later. Haemodilution restores the plasma volume and sometimes the total blood volume through an absolute increase of the plasma volume. Thus the acute anaemia presents as oligaemia and the extent of the red cell loss does not become apparent until subsequent dilution unmasks the earlier loss of red cell mass. Continuing haemorrhage or subsequent infection may contribute to the anaemia and there may be a further disappearance of red cells after primary blood loss has ceased. This may explain the subsequent slight or moderate fall in haemoglobin value after full restoration of the blood loss by blood transfusion. Inadequate transfusion will not restore the red cell mass; haemodilution will still occur and the inadequacy will become apparent by the fall in the peripheral haemoglobin value after transfusion. Reticulocytosis subsequently occurs when blood loss has not been replaced but it is slight or absent after early and sufficient blood transfusion. Bone-marrow observations by the author have shown normoblastic hyperplasia often macronormoblastic in form, indicating that the anaemia is not associated with a depression of erythropoiesis. The erythrocyte sedimentation rate rises partly because of the anaemia and partly because of an alteration in albumin globulin ratio and a raised level of plasma fibrinogen.

Patients with pernicious anaemia

In our experience transfusion of injured patients with pre-existing anaemia, suspected by examination of the peripheral blood before transfusion of being pernicious anaemia, may make subsequent diagnosis difficult. Blood transfusion reduces the reticulocyte response following subsequent administration of vitamin B₁₂ either through restoration of the red cell mass or through the vitamin B₁₂ content of the transfused blood. The vitamin level of the patient's plasma is raised and the cytology of the bone marrow is thereby altered.

Iron and haemoglobin metabolism

Iron and haemoglobin metabolism is complicated by the breakdown of pigment in the injured area and from the proportion of non-viable red cells transfused with stored blood. The serum bilirubin level transiently rises to perhaps 1.0 or 2.0 mg. per 100 ml. and gives an indirect Van den Bergh reaction. The excretion of urinary urobilinogen (including urobilin) is increased and shows two peaks, one within the first 24 hours and the second about a week later. These findings might be explained by a combination of increased red cell breakdown and transient liver dysfunction but Baar and Topley (1956) reported that the increased faecal excretion of stercobilin after injury was related to inadequate blood transfusion. The concentration and total circulating serum iron remained low for a week or longer after injury, returning to normal during convalescence but the fall was less or absent after replacement of blood loss.

Plasma loss

In patients with uncomplicated haemorrhage the volume of lost plasma is limited and is unlikely to produce significant dehydration because even the entire plasma

volume (say 3 l) is only a fraction of the extracellular fluid volume (about 12 l) of which it is a part. Dehydration may occur in hot climates because of loss of water from the skin and after extensive burning because of the loss of large volumes of a plasma like exudate into the burned area (see Sevitt 1957). In crushing injury to limbs and after trauma to body cavities the overall loss of plasma may be greater than that of red cells. The early extravasation is whole blood but the continuing loss is mixed with or composed of inflammatory exudate a protein rich fluid due to tissue damage into the injured limb intestines peritoneal or thoracic cavity. Haemodilution is disturbed by relative haemoconcentration from excess loss of plasma but serial samples of peripheral blood may reveal retarded haemodilution rather than absolute haemoconcentration. Transfusion of whole blood sufficient to correct the loss of red cells may be followed by a haematocrit value above normal.

Plasma proteins

The protein concentration is at first normal and then falls a little as the plasma is diluted with protein-poor interstitial fluid. The fall coincides with that of the haemoglobin value but it is not proportional because of the labile reserves of body protein. Even in very severely injured persons the protein level rarely falls below 5-6 g. per 100 ml. Baar and Topley (1956) compared the protein changes in adequately transfused and under transfused patients and found that the total circulating protein was not low; apparently the low concentrations in the under transfused patients could be explained by increased plasma volume. The albumin globulin ratio is reduced because of a greater loss of albumin than globulin or because of new formation of globulin, or for both reasons. The rise in *alpha*-2 globulin is not related to adequacy of transfusion and is probably newly formed. The plasma fibrinogen level increases and may reach 500-800 mg. per 100 ml some days after injury.

Platelets and clotting factors

The post-operative and post traumatic rise in the blood platelet count is well known. It is maximal 4-8 days after injury and the count may even increase 5-fold or 10-fold. A transient fall often precedes the rising count in patients receiving very large transfusions of stored blood which contains no viable platelets. The battle casualties studied by Scott and Crosby (1955) also showed a temporary depression of plasma prothrombin activity to about 50 per cent of normal for a few days, followed by a second temporary fall, a temporary shortening of the clotting time, and an increased concentration of plasma fibrinogen. There was no increased tendency to haemorrhage or clotting except for a little early capillary oozing after massive transfusion with stored blood whilst post-operative oozing was not excessive. Our own experience is in accord with these findings: occasionally we also find a rapid development of fibrinolytic activity in the plasma, which has disappeared the day after injury. The increase of fibrinogen must be due to new formation by the liver. The fall in the clotting time and the second fall in prothrombin activity are obscure. Scott and Crosby (1955) reported that the latter was not due to a deficiency in labile factor or prothrombin but was often corrected by the addition of heated serum which is free from these factors. After massive transfusion there was some decrease of labile factor probably because of its deficiency in stored blood.

WHITE BLOOD CELLS

Neutrophil leucocytosis

There is a rapid rise in the total white blood cell count after injury owing to a neutrophil leucocytosis but its mechanism is obscure. The response is maximal within 12 or 24 hours and may last 1-4 days; it may merge with or be followed by leucocytosis from subsequent infection or other causes.

Eosinopenia and lymphopenia

The lymphocytes and particularly the eosinophils of the blood fall quickly after injury, reaching minimum values 3-6 hours later. The lymphocyte depression, which is particularly well marked in children, lasts only a day or so and is part of a generalized lymphonecrotic process affecting the lymphoid tissue of the spleen, lymph nodes, Peyer's patches, tonsils and other organs.

Eosinopenia develops quickly after injury, haemorrhage and burns (Hardy, 1950; Laragh and Almy, 1948; Sevvitt, 1951, 1954) and within a few hours the count is found to be falling or has already fallen to a low value, generally less than 20 cells per c.mm. and often zero. The eosinopenia usually lasts between 1 and 4 days, depending on the severity of injury and subsequent operations. It is succeeded by a rise in the count to normal levels or occasionally to a temporary eosinophilic level. This early eosinophilia is to be distinguished from the later eosinophilia which is not uncommon weeks later during convalescence. In very ill patients a low eosinophil count, perhaps after a transient rise, may persist for days; recovery is accompanied or heralded by a rise in the count and death is preceded by a fall to very low or zero levels. Thus the eosinophil count has a prognostic value. Both the lymphopenia and the eosinopenia are related to excessive secretion of adrenal glucocorticoids and the finding of eosinopenia is a useful index of this. The converse is not true: excessive adrenocortical activity may persist after the eosinophil count has risen—an example of the so-called 'escape phenomenon'. There is suggestive evidence that the period of eosinopenia after injury may be shortened by early correction of blood loss by transfusion but the phenomenon is never abolished by restoration of the blood volume. The blood eosinopenia is shortly followed by an eosinopenia in the spleen which may be used as post mortem evidence of adrenocortical hyperactivity (Sevvitt, 1955b).

ELECTROLYTE CHANGES

Severe injury is followed by a considerable fall in the concentration of sodium and chloride in the urine, an increase in the level and total excretion of potassium and a reduction in the flow of urine. The Na/K ratio (in equivalent values) falls to less than unity. These effects may last a few days and are succeeded by sodium and chloride release and a slower excretion of the excess water. The plasma concentrations show little or no change except perhaps a slight fall in sodium and chloride and a slight rise in potassium.

Changes in injured and uninjured tissues

The amount of potassium excreted is greater than can be accounted for by the local injury, since the K/N ratio in the urine is greater than the ratio in tissue

cells. Moreover the decreased excretion of salt and increased excretion of potassium in the face of normal or near normal extracellular (plasma) levels implies a cellular loss of potassium and gain of sodium. Tourniquet trauma in animals has shown that there is an exchange of cellular potassium for sodium in injured muscle (Fuhrman and Crismon, 1951; Macphree, 1955; Tabor and Rosenthal, 1945). The increase of sodium in the injured limb after release of the tourniquet was considerable and was associated with a comparable loss of potassium, a diminution in muscle glycogen, interstitial oedema and an uptake of cellular water. The cation exchange was reversible up to a point but was irreversible after prolonged ischaemia. These changes appear to be independent of adrenal activity as similar changes were found in intact and adrenalectomized rats (Macphree, 1955) and are probably a disturbance, anoxic or otherwise, of the vital activity of cell membranes. After allowing for the amounts in the interstitial fluid Macphree found a slight increase in cellular sodium and a slight decrease in cellular potassium in the undamaged limb. This was not found in adrenalectomized animals and he concluded that cation exchange outside the injured area was a general response to injury associated with functioning adrenal tissue. His results are in contrast to those of Fox and Baer (1947) who in the analogous field of burns found some loss of sodium and gain of potassium in the unburned limb which, they said, pointed to extracellular dehydration and intracellular swelling.

Mechanism

The body mechanisms governing salt retention and potassium excretion are partly related to adrenocortical hyperactivity particularly aldosterone secretion. This hormone promotes sodium retention and potassium excretion and some workers have therefore concluded that its secretion determines the changes in electrolyte excretion after injury. Secretion of aldosterone is independent of pituitary activity but is related to changes in blood volume and to the salt levels of the blood and tissues. Retention of sodium and excretion of potassium after operation occurred in patients subjected to hypophysectomy or bilateral adrenal ectomy when they were maintained on a constant dose of ACTH or cortisone respectively (Graber and Beaconsfield, 1955; Mason, 1955). This and other evidence indicates that adrenal hormones play a permissive rather than a determining role (see page 19) in regulating the renal excretion of electrolytes.

Oligaemia is important in reducing the urinary output and salt excretion. Renal vasoconstriction produces a fall in glomerular filtration rate which is mainly responsible for the decreased flow of urine, but this effect is modified by a decrease in the tubular reabsorption of water (Graber and Sevitt, 1959). The latter suggests that the secretion of antidiuretic hormone is decreased at least periodically after injury. The importance of oligaemia and blood replacement therapy was demonstrated by Flear and Clarke (1955) who showed that post-traumatic retention of sodium and water was considerably less in patients receiving early and adequate transfusion of blood compared with those who were not transfused. This supports the contention of Borst (1948) and Cort (1955) who stressed the importance of blood volume, baroreceptors and nervous stimulation on renal function.

Significance

Salt and water retention may be regarded as a defence mechanism to combat the loss of fluid and salt in the injured area and, through haemodilution, to reduce

oligaemia The free excretion of potassium may counteract the release of potassium in the body and prevent the accumulation of toxic levels in the extracellular fluid. Protection breaks down and hyperpotassaemia develops when potassium cannot be excreted because of oliguric renal failure.

ENDOCRINE RESPONSES

Trauma is followed by an interplay of endocrine activity involving the anterior pituitary gland, both parts of the adrenal gland and possibly the thyroid and other glands. The known results include hypersecretion of adrenaline, noradrenaline, pituitary adrenocorticotrophic hormone (ACTH), adrenocortical glucocorticoids and mineralocorticoids like hydrocortisone and aldosterone respectively. Adrenaline release from fright, pain, injury and other harmful and potentially harmful stimuli was the basis of Cannon's (1915) "flight or fight" reaction. It potentiates sympathetic activity, which is a prominent feature in the injured patient and takes part in the activation of the adrenal cortex, which is essential for homeostasis and survival. Endocrine activity produces, permits or enhances changes in carbohydrate and nitrogen metabolism, the redistribution and excretion of sodium, potassium and water, lymphopenia and tissue lymphonecrosis, blood and splenic eosinopenia and other effects.

Anterior pituitary gland

Activation of the adrenocorticotrophic secretion of this gland is via the hypothalamus, probably through a hormone secreted by the hypothalamus into the hypophyseal portal blood supply (de Groot and Harris 1950; Hume and Wittenstein 1950). The hypothalamus can be stimulated by nervous stimuli coming from the injured area and the afferent reflex path passes through the spinal cord. The adrenal cortex can also be stimulated by intravenous adrenaline, mainly through increasing the production of ACTH (Long and Fry 1945; Vogt 1944), but it is uncertain whether the chief site of action of adrenaline is on the adenohypophysis or on the hypothalamus.

Cytochemical changes in the pituitary glands of patients dying after severe stress have shown a degranulation of the periodic acid-Schiff positive mucoid cells (mainly basophils) (Currie and Symington, 1955a, 1955b). Since ACTH is localized to the basophil cells, it is likely that mucoid-cell degranulation is the morphological expression of ACTH secretion.

Adrenal cortex

The normal resting adrenal cortex contains abundant sudanophil lipid, cholesterol and vitamin C. Experimentally, the secretion of adrenal hormones after stimulation by ACTH, burning or injury is followed by a reduction in the cholesterol content of the gland (Long 1947; Sayers and his colleagues, 1943). A depletion of stainable lipid in the human adrenal gland has been found after severe haemorrhage and in patients dying after injury and burning (Elliott 1914; Sevitt, 1955b; Symington and Davidson 1956) and the ascorbic acid content of the gland falls after various stresses, including burning and injury. The earliest loss of lipid is generally found in the zona reticularis and the inner part of the fasciculata, but sometimes a regular or focal depletion of the glomerulosa also occurs. The inner

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zone loss at first extends evenly but further loss is irregular within the fasciculate. Loss of lipoid from the adrenal cortex follows the eosinopenia in the blood and spleen (Sevitt, 1955b) which indicates that the lipoid material is not the eosinopenic hormone. It is probably a labile store used by cortical cells in synthesizing the active hormones and the ascorbic acid present is probably related to this. This does not mean that complete loss of cortical lipoid can be equated with an exhausted or non-secreting state of the gland. On the contrary, the available evidence indicates that such a gland is hyperactive (see Sevitt, 1957). Post-traumatic adrenocortical hyperactivity is the rule; there is no evidence that hypofunction or failure occurs and there is no basis for therapy with adrenal hormones except occasionally in the special event of post-traumatic adrenal apoplexy (Sevitt, 1955a). The old hypothesis relating traumatic shock to adrenal failure must be abandoned.

Permissive role of adrenal hormones

This concept arose out of the finding that adrenal hormones are necessary but not responsible for the phenomena of sodium and chloride retention, the protein catabolic impulse and gluconeogenesis after injury, burning and other stresses. These reactions do not occur in adrenalectomized or hypophysectomized animals subjected to stress so that the hormones are essential, but when the animal or patient is maintained on a constant dose of cortisone or ACTH respectively the metabolic responses are similar to those which occur in similarly stressed intact animals and men. Without the hormones the biochemical reactions to injury do not occur but they can take place without an increase in their availability. Notwithstanding these limitations the central role of the adrenal cortex in activating certain homeostatic responses after injury is clear.

Posterior pituitary gland

Antidiuretic hormone is released after various stimuli, particularly pain (Theobald and Verney, 1935) and its release might be expected after injury. No direct evidence of this has been obtained and in the analogous field of burns Baar (1956) failed to show antidiuretic activity in the urine. Recent work suggests that there may even be a temporary or periodic suppression of activity since the tubular reabsorption of water by the kidneys is often decreased (Graber and Sevitt, 1959).

METABOLIC DISTURBANCES

Complex biochemical changes involving protein, carbohydrates, minerals, fats and vitamins occur after injury and involve an interplay of nervous and endocrine activity.

Nitrogen metabolism

Loss of flesh after injury is part of folk lore and although Bauer (1872) noted an increase of urinary nitrogen after haemorrhage it was the work of Cuthbertson and his colleagues that opened the field (Cuthbertson, 1929, 1930, 1936, 1942; Cuthbertson, McGirr and Robertson, 1939). After fractures and non-bony injury the urinary output of nitrogen, mainly as urea, increases and is associated with a loss of phosphorus, calcium, sulphur and potassium. The losses can only be partly

explained by muscle atrophy from disuse or lying in bed. The ratios of excess sulphur to nitrogen and excess phosphorus to nitrogen are such that the catabolism must involve general body tissues particularly muscle. This is confirmed by the amount of potassium excreted which is much greater than can be accounted for by local tissue injury. After the initial "shock" phase and during the first week or so the urinary nitrogen rises to a peak and exceeds the nitrogen intake particularly in well-nourished patients (Beattie 1947, Stevenson, Schenhor and Browne, 1945). This catabolic or anti-anabolic phase is associated with an increased basal consumption of oxygen. There is a rough correlation between the amount of nitrogen lost and the severity of injury. The high urinary nitrogen is maintained for 2-6 weeks but gradually declines. The increased excretion of nitrogen is greater than the nitrogen intake and hence the patient is in "negative nitrogen balance". This may be diminished but is never abolished by a high-protein, high-calorie diet during the major part of the catabolic phase. There is evidence that the magnitude of the response can be reduced or postponed by early and adequate transfusion of injured patients (Flear and Clarke 1955) which indicates a close relationship between the maintenance of a normal blood volume and nitrogen metabolism. This is more important clinically than high nitrogen feeding in the early post-traumatic period but after this a high protein high-calorie diet is desirable.

After the catabolic period the urinary nitrogen falls below normal and nitrogen is retained in the body for protein synthesis. The anabolism is so intense that even the products of local proteolysis are used for rebuilding (Striganova, 1940). The cumulative effect of nitrogen loss can be considerable and in improperly treated severely injured persons, may account for losses of up to one third or more of the muscle bulk.

Mechanism and significance

It is known that the urinary loss of nitrogen is accompanied by an increased excretion of adrenocortical steroids, mainly glucocorticoids like hydrocortisone. These hormones can provoke an increased proteolysis and excretion of nitrogen mainly as urea. It was therefore suggested that adrenocortical hypersecretion was the cause of the tissue catabolism (Albright, 1943, Selye, 1946). This view was supported by Campbell and his colleagues (1953) who found experimentally that the increased urinary nitrogen after implanting a 25-mg. pellet of cortisone was similar in value to the effect of a single fracture of the femur and that the effect of implanting 2 pellets was similar to the effect of 1 fracture plus 1 pellet. Nevertheless the present evidence indicates that the protein catabolic response is not determined by an increased secretion of hormones although it does not occur in their absence (Engel 1952, Ingle 1951) so that other factors must also take part or be responsible. The results of thyroidectomy in experimental burns suggests that thyroid activity may be involved (Gribble and Peters 1951, Sellers, You and You, 1950).

Cuthbertson (1942) suggested that the catabolic impulse was a primitive response essential to the survival of a wounded animal unable to hunt for food and that it supplied energy and amino acids for the healing process through metabolizing labile stores of protein. In support of this is the observation that the urinary nitrogen is little increased after injury in undernourished animals presumably the amino acids released after proteolysis are used economically and are not

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available for deamination. Similarly in debilitated patients trauma does not produce an increased nitrogen excretion and the urinary corticosteroid levels are normal or even reduced

Carbohydrate metabolism

The blood glucose level rises rapidly after injury often to hyperglycaemic levels and often produces a temporary glycosuria. The phenomenon is generally short-lived lasting some hours or at most a few days in experimental animals the height and duration of the response are generally related to the severity of injury and the degree of shock produced. In severe injury there is also a rise in plasma lactate and an immediate fall in basal oxygen consumption. There is a reduction in glucose tolerance which may persist for days or occasionally weeks (Thomsen 1938) and there is some evidence of partial resistance to insulin (Howard 1955). Experiment has shown that the immediate source of the raised blood glucose is liver glycogen. The glucose content of the hepatic vein blood in cats subjected to haemorrhage was considerably greater than that in the heart blood, and tying the hepatic vessels before bleeding prevented the hyperglycaemia (Robertson, 1935). The ultimate source of the glucose is partly from muscle glycogen through its breakdown to lactic acid, which after transport in the blood is synthesized in the liver to glycogen by way of pyruvate and partly from gluconeogenesis by the liver from amino acids released after the catabolism of protein in muscle and other tissues. This explains why in animals killed after injury the muscle and carcass stores of carbohydrate are diminished whilst liver glycogen may not be significantly altered.

Mechanism and significance

The breakdown of liver and muscle glycogen is mainly the result of hyperadrenalinaemia whilst gluconeogenesis after protein catabolism is under the influence of the glucocorticoid hormone of the adrenal cortex (Long, 1942) the latter playing a permissive rather than a determining role. Reflex nervous mechanisms may also be involved since the hyperglycaemia after experimental fractures and other limb injuries was said to be prevented by previous section of the nerves to the limb (Ogata, 1936). The central connexions of this pathway are unknown but the efferent flow is probably through the sympathetic system because division of the splanchnic nerves was found to prevent hyperglycaemia after head injury (Mock and de Takats 1929).

There is an apparent contradiction between the destruction of the carbohydrate stores of the body associated with a flooding of the blood and tissues with glucose and the diminished metabolic rate and depression of energy production so characteristic of shock states. The glucose released seems to be inert and glucose utilization is inhibited. Green and Stoner (1954) discussed this paradox and whilst they could not eliminate the possibility that death in shock might result from exhaustion of energy stores in a few vital centres they thought this unlikely. They considered the hyperglycaemic flood to be a defence mechanism whereby the body conserves energy for recovery purposes by incomplete breakdown of carbohydrate in the face of increased demands for carbohydrate in the injured area. The phenomenon does not reflect an exhaustion of the body stores of energy

because there is no significant change in energy rich phosphate like phosphocreatine outside the injured area (Bollman and Flock, 1944) Rosenthal (1943) and others have shown that administration of glucose is of no value to a shocked animal and the same seems to be true for pyruvate and other carbohydrate intermediates. Biochemical studies in this field are far from complete but it is possible that there is an interference with the tricarboxylic acid cycle in shock possibly resulting in a preponderance of glycolysis over aerobic oxidation with a consequent decreased energy production (Engel 1945 Engel Winton and Long, 1943) If this were so the depression of energy production would represent a very primitive protective mechanism. In this context the intimate relationship of oligaemia to the carbohydrate response is uncertain and the effect of the early replacement of blood on the hyperglycaemic impulse would be of great interest.

Mineral and vitamin metabolism

Little is known about calcium and phosphorus metabolism beyond the bare facts that injuries, particularly fractures are followed by a considerable increase in urinary excretion and that the general mobilization results in some demineralization of the skeleton. The plasma calcium level may be normal or slightly raised. The increased urinary calcium excretion has been said to be a potential danger in that calcium might be precipitated and give rise to renal calculi but this seems to be greatly exaggerated.

Saturation tests with ascorbic acid in injured patients have shown a considerable retention of the vitamin (Beattie 1947). Daily supplements of 0.5 g. were not excreted during the first 5-10 days after injury. This indicates a considerable utilization of the vitamin possibly for repair purposes at the site of injury since this vitamin is necessary in the healing of soft tissues and fractures. The connexion between ascorbic acid and the adrenal cortex suggests that part of it is also retained and used for hormone synthesis. Similar changes in ascorbic acid metabolism occur after burning. The healing of incised wounds in previously burned guinea pigs resembles that seen in scurvy and is corrected by administration of large doses of vitamin C (Levenson and his colleagues 1957). If this is true for traumatic injury it indicates that large doses of the vitamin are required to promote normal healing.

There is some evidence that the urinary excretion of thiamine and nicotinic acid are also diminished by injury and if this is confirmed it may also indicate increased utilization and need.

HEPATIC FUNCTION

Some degree of hepatic dysfunction is common after severe injury and is likely to be aggravated by subsequent anaesthesia and surgery but the reserves of liver function appear to be so great that the changes are rarely of clinical importance. Circumstantial evidence indicates that the liver blood flow after trauma is considerably reduced as part of the post-oligaemic regional vasoconstriction. The splanchnic liver circulation is known to be reduced after burning (Dobson and Warner 1954) during general anaesthesia (Shackman Graber and Melrose 1953) and a further reduction occurs during operation. The oxygen content of the hepatic venous blood was found to be normal which in the face of a reduced circulation

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indicates a fall in oxygen consumption. Evidence of liver dysfunction is found in many patients after abdominal and extra abdominal operations the hippuric acid synthesis test is low for days the degree of bromsulphthalein retention is raised and biopsy specimens of liver removed before abdominal closure show some leucocytic infiltration and necrosis of some liver cells (Boyce and McFetridge 1938 Tagnon, Robbins and Nichols 1948 Zamchek Chalmers and Davidson 1949)

Engel (1945) pointed out that the liver was likely to suffer early from a reduced blood flow because the blood supply is mainly venous. Haemorrhage in experimental animals was followed by an early fall of the oxygen saturation in the hepatic venous blood (preceding the fall in the portal blood) which he thought indicated oxygen lack in the liver and explained the early impairment of certain metabolic activities such as the reduced ability to deaminate amino acids and the associated rise in the plasma amino acid nitrogen level. The *in vitro* oxygen consumption by liver slices from animals in a state of shock was reduced in proportion to the height of the amino-acid level in the blood. The liver power to deaminate amino acids was irreversibly damaged by a period of anoxia of duration longer than 45 minutes and Engel concluded that the liver was particularly susceptible to oxygen lack.

Jaundice

The occurrence of hepatic jaundice after blood transfusion in some injured and burned persons (Sevitt, 1958) suggests that some degree of liver dysfunction is not uncommon after many injuries. Jaundice and bilirubinuria appeared shortly after transfusion with stored blood but there was no evidence of blood incompatibility or haemolytic reaction. The jaundice was associated with a direct serum van den Bergh reaction urobilinuria often a raised serum alkaline phosphatase level and sometimes increased thymol and zinc sulphate serum turbidity reactions. It resulted from a combination of temporary hepatic impairment and acute bilirubin loading. The former was related to a reduced hepatic circulation following injury subsequent operations and anaesthesia, singly or in combination, whilst the acute loading of the blood stream with bilirubin was largely derived from the extravascular destruction of the non viable population of red cells in stored blood.

In other words transfusion acted as a bilirubin loading test of liver function. Many of the battle casualties studied by Scott, Olney and Howard (1955) also had a raised serum bilirubin level after blood transfusion (but neither jaundice nor bilirubinuria were reported) and evidence of liver impairment namely abnormal retention of bromsulphthalein increased urinary excretion of urobilinogen some loss of plasma prothrombin activity and a raised serum cephalin flocculation.

TRAUMATIC URAEMIA AND RENAL TUBULAR NECROSIS

(See also Chapter 7)

The essential pathogenesis of traumatic uraemia is a persistently low rate of glomerular filtration at least partly due to renal ischaemia (Graber and Sevitt, 1959 Sevitt, 1959) (see Fig. 19 Chapter 7). Prolonged renal ischaemia also produces tubular necrosis but traumatic uraemia and tubular necrosis may occur independently. Common pathogenetic factors explain the frequent association. This

is in conflict with the prevailing view that tubular necrosis is the pathological basis of acute renal failure. The condition is uncommon in patients adequately transfused shortly after injury so that correction of oligæmia protects against renal failure after trauma. Most cases now seen in an efficient traumatic unit follow either abdominal injury where vomiting, paralytic ileus and other complications play a part or head injury, or occur at a later stage after certain complications, particularly pulmonary embolism.

Traumatic uræmia is either non-oliguric or severely oliguric but the glomerular filtration rate is reduced in both kinds and is responsible for the severe azotæmia. Severe oliguria is particularly dangerous because of water retention, hyperpotasæmia and acidosis which are due to the inability to lose water, potassium and the acid end products of metabolism. The non-oliguric form is easily overlooked unless the possibility is recognized and regular blood urea estimations or other tests are carried out. The volume of urine passed is determined by the balance between the filtration and tubular reabsorption of water. Oliguria is absent when a decrease in tubular reabsorption balances the low filtration and is present when it fails to compensate.

Glycosuria is absent, sodium and chloride are retained as in injured patients without uræmia, whilst the excretion of potassium is free and limited only by the volume of urine passed. Thus the composition of the urine indicates continued tubular function. The apparent paradox between tubular necrosis and tubular activity indicates the presence both of damaged and functioning nephrons and is an index of kidney tissue reserve.

REFERENCES

- Albright, F. (1943) *Harvey Lect.* 38, 123.
 Baar Stella (1956) *J. clin. Path.*, 9, 144.
 — and Topley Elizabeth (1956). *Acta med. scand.*, 153, 319.
 Bauer J. (1872) *Z. Biol.* 8, 567.
 Bayliss, W. M. Cannon, W. B. Frazer J., Hooper A. N. and Corwell, S. R. (1919) In "Wound Shock and Haemorrhage." *Spec. Rep. Ser. med. Res. Coun., Lond.*, No. 25.
 Beattie, J. (1947) *Brit. med. J.* 2, 813.
 Bollman, J. L. and Flock, E. V. (1944). *Amer. J. Physiol.* 142, 290.
 Borst, J. G. G. (1948) *Acta med. scand.*, Suppl. 207.
 Boyce, F. F., and McFetridge, E. M. (1938) *Arch. Surg., Chicago* 37, 443.
 Campbell, R. M. Sharp G. M. E. Boyne, A. W., and Cuthbertson, D. P. (1953) *Nature Lond.* 172, 158.
 Cannon, W. B. (1915) *Bodily Changes in Pain, Hunger, Fear and Rage* New York: Appleton.
 Cort, J. H. (1955). *Physiol. Bohemoslovenica*, 4, 14.
 Courmand, A., Riley R. L., Bradley, S. E., Breed, E. S., Noble, R. P., Lanson, H. D., Gregersen, M. L., and Richards, D. W. (1943) *Surgery* 13, 964.
 Crosby, W. H. and Howard, J. M. (1954) *Blood* 9, 439.
 Currie, A. R. and Symington, T. (1955a). In *Ciba Colloquia on Endocrinology* Vol. 8, p. 396. London: Churchill.
 — — (1955b) *Proc. R. Soc. Med.* 48, 908.
 Cuthbertson, D. P. (1929) *Biochem. J.*, 23, 1328.
 — (1930) *Ibid.* 24, 1244.
 — (1936) *Brit. J. Surg.* 23, 505.
 — (1942) *Lancet* 1, 433.
 — McGirr J. L. and Robertson, J. S. M. (1939) *Quart. J. exp. Physiol.*, 29, 13.
 Dacie, J. N. and Homer G. F. (1946) *Lancet* 1, 371.
 Dobson, E. L., and Warner G. F. (1954). *Fed. Proc.* 13, 36.

REFERENCES

- Elliott, T R. (1914) *Quart J Med* 8, 47
 Engel, F L. (1945). *J Mt Sinai Hosp* 12, 152.
 — (1952) *Endocrinology* 50, 462.
 — Winton, M G and Long, C N H (1943) *J exp Med.*, 77 397
 Fine, J (1952) In *First Conference on Shock and Circulatory Homeostasis* Ed. by H D Green. New York J Macey Foundation.
 Fear, C T G and Clarke, R. (1955) *Clin Sci.*, 14, 575
 Fox, C L., and Baer H (1947) *Amer J Physiol.*, 151 155
 Fuhrman, F A., and Crismon, J M (1951) *Amer J Physiol.*, 167 289
 Graber I G., and Beaconsfield, P (1955) *Brit med J.*, 2, 704
 — and Sevvit, S. (1959) *J clin Path* 12, 25
 Grant, R. T., and Reeve, E. B (1951). "The General Effects of Injury in Man." *Spec Rep Ser med Res Coun., Lond.*, No 277
 Green, H. N (1943). *Lancet* 2, 147
 — and Stoner H B. (1950) *Brit med J* 1 805
 — (1954) *Brit med Bull.*, 10, 38
 Gribble M G., and Peters, R A. (1951) *Quart J exp Physiol.*, 36, 119
 de Groot, J and Harris, G W (1950) *J Physiol.*, 111 335
 Hardy J D (1950) *Ann Surg* 132, 189
 Howard, J M (1955) *Ann Surg.*, 141 321
 — Frawley J P., and Artz, C. P (1955) *Ibid* 141 337
 Home, D M., and Wittenstein, G J (1950) In *Proceedings of the First Clinical ACTH Conference* Ed. by J R. Mote. Philadelphia Blakiston.
 Ingle, D J (1951) *Recent Progr Hormone Res.*, 6, 159
 Larrigh, J H., and Almy T P (1948) *Proc Soc exp Biol., N Y.*, 119 499
 Lauson, H. D., Bradley S E., and Courmand, A. (1944) *J clin. Invest.*, 23 381
 Levenson, S M., Upjohn H L. Preston, J H., and Steer A. (1957). *Ann Surg.*, 146, 357
 Long, C. N H (1942) *Endocrinology* 30 870.
 — (1947) *Recent Progr Hormone Res* 1 99
 — and Fry E. G (1945). *Proc Soc exp Biol N Y.*, 59 67
 McMichael, J (1944) *J Amer med Ass.*, 124, 275
 Macphoe, I W (1955) *Clin. Sci.*, 14, 451
 Mason, A. S. (1955). *Lancet* 2, 632.
 Mock and de Takara (1929). Quoted by Thomsen, V (1938).
 Noble, R. P., and Gregersen, M (1946) *J clin Invest.*, 23 381
 Opals, T (1936). *Arch. klin Chir.*, 187 19
 Richards, D W (1943). *Harvey Lect.*, 217
 Robertson, J D (1935) *J Physiol.*, 84, 393
 Rosenthal, S. M (1943). *Publ Hlth Rep., Wash.* 58, 1429
 Sayers, G., Sayers, M A. Fry E. G., White, A., and Long, C. N H (1943) *Yale J Biol Med* 16, 361
 Scott, R., and Crosby W H (1955) *Ann Surg* 141 347
 — Olney J M., and Howard, J M (1955) *Battle Casualties in Korea* Vol. I p. 149 Washing ton U.S. Armed Forces Report.
 — Howard, J M., Shorr E., Lawson, N., and Davis, J H (1955) *Ann. Surg.*, 141 504
 Sellers, E. A., You, S S., and You, R. W (1950) *Endocrinology* 47 148.
 Selby, H. (1946) *J clin. Endocrin.*, 6, 117
 Sevvit, S (1951) *Brit med J.*, 1 976.
 — (1954) *Ibid.*, 1 541
 — (1955a). *J clin. Path.*, 8, 185
 — (1955b). *J Path Bact* 70, 65
 — (1957) *Burns Pathology and Therapeutic Applications* London Butterworth.
 — (1958) *Brit J Surg.*, 46, 68.
 — (1959). In the press.
 Shackman, R., Graber I G and Melrose, D G (1953) *Clin Sci.*, 12, 307
 Sheehan, H. L. (1948) *Lancet* 1 1
 — (1951). *Circulation* 3, 42.
 Shorr E., Zwerbach, B W., and Furchgott R. F (1948) *Ann N Y Acad Sci* 49 571
 — — (1951). *Circulation* 3, 42.
 Steinman, J A. P., Schenbor V and Browne, J S L. (1945) *J Canad med. Serv.*, 2, 345

- Striganova A (1940) *C. R. Acad Sci U.R.S.S.*, 27 385
- Symington, T and Davidson, J N (1956) *Scot med. J.*, 1 15
- Tabor H., and Rosenthal S M (1945). *Publ Hlth Rep Wash.*, 60, 373 401
- Tagnon H J., Robbins, G F., and Nichols, M P (1948) *New Engl J Med.* 238, 556.
- Theobald G W., and Verney E. B (1935) *J Physiol.*, 83, 341
- Thomsen, V (1938) *Acta med scand Suppl* 51
- Topley Elizabeth, and Clarke, R (1956) *Blood* 11 357
- Vaughan, Janet (1948) *Brit med J* 1 35
- Vishnevsky A. V (1941) *Arch Sci Biol., Moscow* 62, 3 Reviewed in *Bull War Med.* (1943), 4, 67
- Vogt, M (1944) *J Physiol* 103, 317
- Whitely H J and Green H N (1952). *J Path. Bact* 64, 224
- Wolfson, J (1957) *Brit J clin Pract.*, 10, 783
- Zamchek, N., Chalmers, T C. and Davidson, C. S (1949) *Amer J Med.*, 7 409

CHAPTER 3

PRIMARY ASSESSMENT OF THE INJURED PATIENT

RUSCOE CLARKE

THE EARLY diagnosis and treatment of injuries warrant particular consideration. Problems arise because injured patients arrive at all times of the day and night, often in numbers and at major centres sometimes in a continuous stream. Such patients differ from those with many other ailments not only by presenting as emergencies but because the event of injury provides a known starting point for the development of pathological disturbances. The patient may be seen before such changes are evident and the minimum of physical signs may be combined with the maximum of danger. On the other hand patients who have sustained organic ally trivial injuries may be frightened by the events of injury and appear seriously ill as a result.

A latent period between onset of pathological changes and clinical effects is usual in patients suffering from a variety of disorders including infections, de generative conditions, endocrine or nutritional disturbances and malignant neoplasms. The onset of the pathological state may be difficult to date and the medical problem arises only when the effects have reached beyond a certain point. In contrast to this the injured patient is frequently seen before clinical effects have developed or when they are only in the early stages. Those responsible for the care of the injured are therefore given an opportunity to anticipate a range of harmful sequelae, most of which can be prevented more easily than cured. An obvious example is wound infection. Preventive treatment may be simple. Its neglect can lead to septicaemia, gas gangrene and death. More dramatic still is the use of transfusion, not only to cure states of "traumatic shock" but to prevent the development of such disturbances.

SUCCESSION OF PATHOLOGICAL EVENTS

The processes initiated by trauma may lead to progressive deterioration unless reversed in time and sometimes reversal is possible only by definitive therapy. The processes are general as well as located at the site of injury. Local and general effects for purposes of classification may be considered separately but in life they are interrelated and this must be appreciated in clinical practice. There is a variety of possible effects from an injury to any region of the body because a variety of organs and tissues may be involved whilst multiple injuries increase the range of effects. A knowledge of common processes and mechanisms of injury may limit the permutations to be considered but the variety remains. Fortunately the complicated damage may sometimes be associated with clinical and other evidence from which a more or less detailed anatomical diagnosis can be made. Even when this is not possible at a first examination it may become so

in time to prevent serious effects. The importance of anatomical diagnosis must never be overlooked.

The difficulties inherent in diagnosis are increased when facilities for examination, investigation and treatment are inadequate, or when responsibility for primary decisions devolves on inexperienced junior members of the hospital staff. The training of the young doctor in routine ward diagnosis and after-care is easier than equipping him for a high standard in the care and management of the acutely injured patient. The problem is particularly difficult when inadequate facilities are combined with insufficient and inadequately trained staff and too many patients.

An important feature of the initial handling of injured patients is that diagnosis is a process and may not be completed in a single stage. A preliminary history may lead to preliminary examination. A primary assessment must often be made before either history or examination is completed. Essential (first aid) treatment of wounds, bleeding, or other urgent matters may be needed before investigation is systematically begun. Surgery itself is often part of the process of investigation, and all treatment including transfusion is part of a clinical trial leading to more detailed diagnosis. Diagnosis is not just a matter of labelling but of arriving at a mental picture of the nature and extent of injury, the abnormal processes that have been set in motion as a result, the present state of the local and general reaction of the patient, and what is likely to happen next, with or without treatment. An early prognosis often has to be made and some of this assessment may have to be shared with the patient or his relatives.

The common important sequelae are haemorrhage, infection and their effects. Severe local injuries can produce major disturbances of the circulation and in older patients coronary thrombosis is possible and later thrombo-embolism is common. Other patients suffer through the reaction of the nervous system to injury and to circumstances associated with injury so that there is a complex combination of neurogenic effects and psychological overlay. The primary impact of injury provokes a complex series of endocrine responses which may be influenced by subsequent events.

For purposes of description a regional classification of injuries is necessary: injuries to the head, neck, chest, abdomen, pelvis, shoulder, arm, forearm, hand, thigh, leg, foot, and so on. Regional classification may guide the clinician on the kind of disorder likely to result from local injury. The relationship between anatomical structure and physiological function means that the nature of the local injury may determine the nature of systemic complications. An injury to the gut can produce systemic illness and a threat to life from leakage and peritonitis. A tear of the gut sutured incorrectly can produce a stenosing scar and intestinal obstruction. A wound of the neck can cause fatal haemorrhage from major vessels, laryngeal obstruction from oedema, bronchopneumonia from inhaled blood, or a dropped shoulder from a cut accessory nerve. Immediate and later effects need to be considered in relationship to the anatomical site of injury and these can result as much from the process of repair as from the nature of the injury.

TYPES OF INJURY

The anatomical questions—what structures have been injured, where, how much, and in what way—must be answered and the answers may be vital for treatment.

TYPES OF INJURY

and prognosis. With major open wounds it is sometimes possible to inspect the extent of damage at a glance or with but little incision or retraction during surgical exploration. Other wounds combine a small breach of the surface with gross internal damage which is only disclosed by surgical incision.

Minor wounds may be fully revealed at first inspection or subsequent operative cleaning, but the obvious wound may not be the main lesion and honest patients can give very misleading histories.

A 35-year-old worker caught her finger in a roller and fractured the lower end of her radius. Primary treatment of the presenting injuries was carried out at hospital. Subsequently she complained of pain in the shoulder and back and gave a history that when her finger was trapped in a moving belt she had been twisted right round and flung down on her back. On first attendance interest was focused on her bleeding finger and painful wrist.

Repeatedly it is found that the first story is coloured by the apparent extent of injury yet in many injuries the information derived from inspection is limited and much assistance can be gained from a detailed history particularly when this is related to clinical signs.

Without attempting a comprehensive survey it is worth considering some problems of anatomical diagnosis in relation to particular types of injury.

Penetrating wounds

Penetrating wounds from weapons are uncommon in Britain, but they are particularly important because of the extent of injury that can be associated with a trivial external wound.

A young man was engaged in a brawl. He did not appear to have been hurt until on his way home he remarked that he must have scratched his side. His friend persuaded him to go to hospital where he was found to have a minute wound over the right costal margin such as might have been inflicted by a thin dagger. A junior medical officer put a stitch in the wound, detained the patient and ordered a pulse chart to be kept. There were no physical signs and no other symptoms. Next morning the patient collapsed and was found to have a rapid pulse, low blood pressure and a rigid abdomen. At exploration the wound was shown to have penetrated the pleural cavity, diaphragm, liver, gall bladder, ampulla of Vater, anterior and posterior walls of the duodenum, and finally the pancreas! The patient recovered.

A small child presented with a tiny wound of the abdomen from a penknife. This was explored and found to track for 2 inches. Next morning the child collapsed and died. At autopsy there were six perforations of the small intestine. The hole in the posterior rectus sheath and peritoneum was 3 inches from the skin wound. The discrepancy was explained by the doubled-up position of the body when the knife entered.

Penetrating wounds from sharp and narrow objects can be relatively straightforward anatomically because damage is likely to be confined to a narrow track the direction of which can be ascertained. Penetrating wounds inflicted with knives, daggers, spikes of railings, nails, sharp pieces of wood, darts, arrows and so forth must always be considered in terms of damage in the depths. Even with the sharp object still in place it is not always easy to determine the likelihood of deep damage. Penetrating wounds of the intestine may produce no physical signs for many hours. A stab wound of the femoral artery may be associated with no external

bleeding and little swelling. A dart can penetrate the skull of a child and perforate the dura without producing any disturbance of consciousness or behaviour (Fig 2). A 1½-inch nail can penetrate the tissues of the abdomen as far as the psoas muscle without anything being clinically evident other than the wound of entry. Yet such injury can be fatal. Sometimes the direction of the track is clear but it is often necessary to imagine a variety of possible directions. Ambroise Paré insisted on the value of ascertaining the position of the patient at the time of injury and an appreciation of this can make sense of an otherwise bizarre combination of lesions.



FIG 2.—Penetrating wounds can be dangerous—even a dart can lead to fatal haemorrhage or meningitis. The child shown here recovered uneventfully after exploration.

Once the general direction of a penetrating wound is clear it can often be known that certain organs and structures have not been damaged. A small fragment of metal that has traversed the right chest wall and lodged in the right upper quadrant of the abdomen can be shown radiologically to be in the liver. Clearly it has not traversed the general abdominal cavity and laparotomy may not be indicated. The importance of reaching such conclusions from clinical and radiological evidence must be considered in relationship to the ease or difficulty of carrying out adequate exploration of the wound track. A nail or screwdriver which has pierced the hand may be treated conservatively if detailed exploration would involve a complex dissection. Exploration of a smaller track in the vicinity of the knee joint would be justified because full exploration should do no harm.

Penetrating wounds in civilian practice are usually single or limited in number except in the case of shotgun pellets and boiler explosions. The former may be widely scattered but are rarely dangerous. The latter may be associated with extensive deep damage and resemble war wounds from mines and mortars. When such wounds are multiple there is always a danger that one or more may be missed, and those the most dangerous. The risk of missing one of a number of multiple wounds is particularly great when the patient is seriously injured and the known wounds appear to explain the clinical picture. Whenever multiple lesions are

TYPES OF INJURY

present or seem likely from the nature of the injury it is essential to strip the patient completely so that the whole body front and back can be thoroughly and systematically examined. Perhaps the most dangerous penetrating wounds are those of the buttock which are easily missed and can involve abdominal or pelvic organs, nerves or vessels. It is also easy to miss wounds at the back of the knee.

A 54-year-old man involved in a road accident reached hospital 12 minutes later "gravely shocked", restless, and irritable with closed fractures of the pelvis, both femora, and a large inguinal haematoma. He was considered to be grossly exsanguinated but transfusion through the cephalic vein was of little benefit. Arterial transfusion failed and he died 45 minutes after admission. He had been stripped of his blood-stained clothing and in the shock room no blood was seen near the knee. Autopsy revealed a 3-inch laceration behind the knee which had severed the popliteal vessels. Death was due to air embolism.

Limb wounds

Penetrating wounds of limbs, and especially the roots of the limbs, thigh, groin and clavicular regions must always be considered in terms of possible damage to main vessels and nerves.

A young infantryman received a tiny wound of the shoulder. On examination he appeared to have an obscure paralysis of the hand and "glove" anaesthesia. He was evacuated with the diagnosis of "penetrating wound of the axilla, hysterical paralysis of the hand". At exploration he was found to have a complete section of the musculocutaneous nerve, hemidivision of the median nerve, bruising of the ulnar nerve, and a lateral wound of the brachial artery and one of its venae comites. Exploration enabled the arterial wound to be sutured and the nerve injury to be diagnosed.

Accidental damage to main blood vessels from penetrating wounds (notoriously those sustained from a butcher's knife) either bleed profusely and are obvious or bleed very little and can be missed. A penetrating wound alongside the main vessels must always be suspect. The superficial femoral vessels can be divided without subsequent swelling or interference with the circulation distal to the wound. Occasionally such lesions are associated with a pulsating haematoma and a bruit on auscultation. Early diagnosis is important because such lesions are easily repaired or grafted. Diagnosis must not be made by poking around in the wound, and even severe bleeding must first be dealt with by pressure. The blind use of forceps increases damage to vessels and may make suture impossible. Such wounds should be explored under ideal conditions and if significant bleeding is absent the patient should be transferred to a centre where full facilities exist. The journey can be made safe by the application of firm bandages over plenty of cotton wool.

Blood loss is considered elsewhere but the immediate care of the open wound involves assessment of bleeding of various categories and degrees of severity.

Wounds of head and abdomen

Penetrating wounds of the head and abdomen are of special importance, first in relation to the risk of infection, sometimes on account of the effects of bleeding. Both these factors are relevant in chest wounds although here it is the effect on vital organs that needs chief consideration. The heart may be involved directly and any such suspicion calls for careful assessment, caution, and expert advice.

More frequently it is the lungs that are interfered with and this can happen in a number of different ways, from dramatic disturbances of respiratory function with open wounds of the pleura to the more insidious effects of closed injuries.

The major wound of a limb or the trunk involving muscle and bone is dangerous primarily from the extent of bleeding, less dramatically from the risk of severe infection particularly gas gangrene where major muscle masses are severely damaged.

Closed Injuries

Closed injuries are less obviously dangerous than bleeding open or penetrating wounds, and the need to establish an early diagnosis and to proceed with treatment is insufficiently appreciated. The importance of early diagnosis and treatment is made clear by considering closed injuries as wounds involving a variety of tissues excluding only the skin. Even the skin may be damaged although it appears intact, whilst its blood supply may be threatened from the extent or sequelae of the injury.

Closed wounds result from various types of violence: direct blows, acceleration or more frequently deceleration, bending, twisting, crushing, tearing, and various combinations of these. The extent of injury is often more widespread than with open penetrating wounds. In falls from a height and particularly in injuries from vehicles of all kinds (on roads, railways and in industry) multiplicity is the common feature. The more violent the injury the more likely is the skin to be broken or damaged somewhere so that some element of the total injury is in fact an open wound or fracture. This feature, however, does not necessarily affect the urgency of diagnosis and treatment: this depends on other features, particularly major fractures, extensive tissue damage, or damage to the vital contents of the cranial, thoracic or abdominal cavities.

The major risk of the open wound is direct infection, but there are many closed injuries where infection is likewise a major hazard.

Concealed open wounds

Apparently "closed" injuries may open into internal body cavities so that they include breaches of surfaces just as open to infection as the more obvious skin wound. Injuries of the intestinal tract provide a dramatic example but any breach of surface of the alimentary or respiratory tracts is an "open wound".

Every fractured skull with blood or cerebrospinal fluid coming from the ears, nose, or throat must be considered as an open wound communicating with the inside of the cranial cavity. Every fracture of the skull involving the middle ear, mastoid air cells, frontal ethmoid, sphenoid, or maxillary sinuses is an open fracture and carries the risk of meningitis (see Chapter 16). Every chest injury with surgical emphysema, pneumothorax, or haemoptysis is an open wound of the lungs.

Closed injuries of the bile passages, liver, pancreas or urinary tract are less directly "open" but allow leakage of irritating fluids which predispose to infection unless properly treated.

Such injuries or the mere suspicion of such injuries calls for alertness, careful investigation and prophylactic treatment which may range from observation and antibiotics to surgical exploration.

Indirect wound infection

The diagnosis of a closed injury is often regarded as removing the danger of wound infection except by the further and perhaps rare "accident" of bacterial contamination via the blood stream. It is often overlooked that even when skin and mucous membrane are primarily intact, injury may produce effects which subsequently interfere with their vitality.

Damaged skin or mucous membrane may allow the direct passage of micro-organisms. This is particularly true of the alimentary tract and experimental work suggests that severe haemorrhage may interfere with the blood supply to the gut sufficiently to allow intestinal flora to reach the liver in greatly increased numbers (Fine and his colleagues, 1952).

It is quite common for skin which has at first appeared intact to die later. This can lead to a demarcated hard dry slough with a slowly developing plane of cleavage and a minimal inflammatory response but the skin has been breached and an underlying closed fracture may become evidently open. Sometimes the skin sloughs more rapidly with early infection and a systemic response. Such moist sloughs require urgent excision and again the wound is no longer closed. The late appearance of skin death may be the direct result of the original injury: it may follow spreading local thrombosis of arteries or veins or it may be due to increasing tissue tension leading to interference with the blood supply. Sometimes the skin damage may result from a secondary injury from within in the case of an inadequately splinted fracture exerting pressure on the overlying skin from without when friction or pressure is present from an overlying plaster or sharp turn of bandage.

Similarly a damaged area of the intestinal or urinary passages may be the site of a delayed rupture: the original damage, possibly incomplete, having predisposed to necrosis of the remaining wall with a final rupture probably due to increase in pressure within the lumen.

When large tissue masses are devitalized or a haematoma is allowed to accumulate in the depths, infection can reach the resulting culture medium via the blood stream, the lymphatics or directly by invasion from neighbouring parts. Intact skin does not exclude the risk of infection. On the other hand, when the skin is breached restoration of skin continuity by suture or grafting is the best defence against added infection so long as the underlying tissues are viable and dead space is obliterated.

Finally open surgery for the repair of the closed wound itself carries a risk of added infection, which should normally be small compared with the alternative risks that operative intervention is designed to avoid.

Other sequelae

Emphasis has been placed above on the risk of infection in closed injuries but the more common result of severe closed injuries is bleeding, which is discussed in Chapter 5. In brief all significant closed fractures and soft tissue injuries are associated with vascular damage and bleeding into the tissues the extent of which may determine the severity of the general reaction of the patient. This may be overlooked in patients with multiple injuries if the attention of both patient and medical staff is focused on particular local injuries. Progressive bleeding from a

fractured pelvis or ruptured spleen may continue with minimum symptoms or signs right up to the time of sudden and dangerous collapse

Two other conditions namely fat embolism and pulmonary thrombo-embolism require mention particularly as their frequency and significance has not in the past been adequately recognized. Pulmonary fat embolism almost invariably follows bony injury and results from the liberation of marrow fat into the venous circulation and thence to the lungs, but it is rarely if ever of clinical significance. Systemic fat embolism is less frequent but more important in practice and is sometimes lethal through severe involvement of the brain (see Chapter 14)

Thrombosis of deep veins in the lower limbs is very frequent in middle aged and elderly patients at rest in bed from any cause including injury its major complication is pulmonary embolism which is often fatal (see Chapter 17)

Provisional diagnosis

In the early assessment of the injured patient the risk or the cause of systemic reactions must often dictate primary diagnostic measures and treatment but when the risks of infection or bleeding are slight and particularly in the younger age groups the primary concern must be the nature and extent of local damage and its significance for the particular patient. The first consideration in most relatively minor injuries to the head and trunk is that of excluding serious complications. In limb injuries careful clinical examination may be needed to elicit the presence of small local lesions which can be decisive in their functional importance. Early radiological examination is frequently essential to exclude bony damage. It will reveal the presence or absence of major bony damage or dislocation, and their presence may indicate that certain ligaments, periosteum, muscles, or other structures may or must have been damaged. Taken in conjunction with detailed clinical examination it can lead to a considerable amount of knowledge of the anatomical lesion. In moderately severe injuries this may allow treatment to proceed, although further information will result from exploration when surgery is indicated. In less serious injuries without major fractures a detailed diagnosis may be difficult although still important, at the acute stage. Once major bony damage has been excluded the patient can usually be calmed sufficiently to allow systematic and detailed clinical examination to establish the normality or otherwise of all important structures. Such examination may be particularly valuable early that is, before swelling has developed. At times the presence of swelling may make it difficult to obtain detailed information which only becomes available later as swelling subsides and movements become possible. In patients with closed injuries to joints, muscles and tendons, repeated observations will frequently provide information not forthcoming from a single investigation. The clinical course will provide information of the presence or absence of certain types of important injury.

Even when the skin is intact and infection unlikely early operation (within a matter of hours rather than days) is often the optimal treatment. Early operation facilitates the diagnosis of deep damage and often provides the best opportunity for repair. Late exploration may be made difficult by developing scar tissue while the anatomy of injury may be concealed by the process of healing. Early surgery depends on definite indications but is never possible unless it is considered when the patient is first seen.

CLINICAL SORTING

Even in more severe injuries, when life-saving considerations predominate it is often possible to plan reparative surgery and the patient's return to as near a normal life as the extent of injury permits.

Crushing injuries

Some of the literature would suggest that there is a particular type of injury that can lead to "the crush syndrome". Interest in the syndrome of renal failure following injuries that appeared to be progressing satisfactorily was aroused by Bywaters and Beall (1941) when they studied this syndrome as a complication of injuries produced by falling masonry during the air raids in Britain. Most of these patients had in fact been trapped by the rubble so that their limbs remained crushed in addition to being damaged by the fall. There is certainly a type of injury affecting the limbs, and sometimes the trunk due more to the duration of pressure than to the impact of the initial blow. It is characterized by gross swelling and blistering of the skin, which may appear as if it has been burnt. Such patients can lose plasma out of proportion to whole blood and may present in a state of severe oligæmic shock. Recovering from this or even if they appear to be fit from the start, they may subsequently develop renal failure.

It is now recognized that the renal failure is not specific to crush injuries (see Chapter 7). The injuries themselves are not necessarily "pure" the swollen limb has usually sustained direct damage so that tissues are torn as well as crushed major blood vessel damage can be present in addition to fractures of the limbs and trunk. The author remembers a patient buried by masonry who developed renal failure but who had not been crushed. He was simply trapped and had been lying for 18 hours with one knee fully flexed obstructing the venous circulation. This particular patient resembles the animals where "crush syndrome" is produced by a tourniquet.

CLINICAL SORTING

The handling of patients in a busy casualty department requires a process of triage and the principles underlying sorting are also relevant to the handling of individual patients at a small hospital or in a general practitioner's surgery.

It is not sufficient to rely on surface appearances for a decision on the degree of severity of injury particularly when the injury is recent. The frightened patient with a trivial injury may present with death like pallor an almost imperceptible pulse and apparently no blood pressure. The patient with potentially lethal injuries may walk into hospital looking the picture of health. A brief history and examination may establish that the injury is local and minimal. This conclusion can often be reached from an account of the accident given by the patient or bystanders particularly in industrial or domestic injuries. If the history is of a fall downstairs or from a height or of any accident where vehicles are involved, multiple injuries should be suspected. The patient with known multiple injuries or complaining of a single injury to the head, trunk or lower limbs must lie down for examination. It is only the patient with an upper limb injury or with trivial injuries to face or trunk who can be examined sitting or standing. Even so many patients with wounds or fractures of the upper limb may faint on removal of bandages or examination of a fracture. Lying down is safer if couches are available.

Most injured patients when first seen in the casualty department will either

appear to be clinically well or to be suffering from some degree of so-called traumatic shock. Two other types of clinical disturbance requiring a different approach at primary examination are first disturbance of consciousness, typically from head injuries and second disturbance of respiratory function from injury to the chest or respiratory passages.

The accident may be sometimes only incidental. Head injuries can result from a fall due to some medical cause or to alcohol without the head injury being any the less genuine or important.

Assessment of the ill patient

The injured patient who seems to be systemically ill should be nursed flat except when there is respiratory difficulty when it may be found that he is better sitting up. There is a widespread tendency to keep injured patients in the position in which they reach hospital. This may be wise in the case of chest injuries and suspected spinal injuries, but it is important to find out from ambulance men, first-aid workers or others with the patient just why any particular position has been adopted. There may be no reason at all. An early medical decision must be made on how much the patient can be moved for undressing and examination and whether any particular position is essential or desirable.

The next decision involves the degree of urgency of examination. The majority of patients can give routine details of age, sex, history and so on before medical investigation begins and the taking of a medical history can precede examination. In some patients the injuries are so obviously severe or are associated with a clinical state that appears so desperate that a preliminary medical examination should precede removal from the stretcher. Much can be learnt in a few seconds from the following observations: (a) the degree of unconsciousness, alertness or anxiety; (b) the response to a simple inquiry; (c) the colour and feel of the skin of the face; (d) the nature of respiration; (e) the presence and quality of the pulse; and (f) the history and diagnosis given by those bringing the patient to hospital.

A number of injured patients reaching hospital *in extremis* can be saved if blood transfusion is started within 5-10 minutes. The decision can often be taken within a minute or two of the patient's arrival provided that an experienced doctor is available. The most dangerous delays commonly occur first before any doctor sees the patient, and secondly when the reception doctor is both inexperienced and not supported by an organization which makes a more experienced opinion available in a few minutes and blood rapidly available for transfusion.

When there are no signs of continuing bleeding, the patient who is conscious, breathing regularly and has a palpable pulse can always wait a few minutes for careful transfer to a couch, the taking of a basic history and at least the first stage of undressing before examination begins.

The next thing to be sought for is evidence of extensive injuries or an important single injury. The patient with a normal blood pressure may have had a severe haemorrhage but if the accident has been very recent he can wait 20 minutes for primary assessment provided that he is watched for deterioration. Even before this it may be clear that transfusion is required, so that blood can be taken for grouping and cross matching.

The better the clinical state of the patient, the less severe the injuries detected

and the less time that has elapsed since injury the more time there is available for systematic history taking followed by clinical and other investigations. The more severe the injuries and the patient's general condition and the longer the interval from the accident the more it is essential for a preliminary investigation to be completed rapidly so that a plan for emergency treatment can be made. The greater the injury the greater the need for quiet calm deliberation and this is consistent with efficient speed. The bigger and the more complex the injury the greater is the need for a team approach with delegated individual responsibilities. It is often necessary to have one person responsible for the airway and another for the introduction of a transfusion needle. In severe multiple injuries a team of 2 or 3 doctors and half a-dozen nurses is often the bare minimum required.

Detailed indications for transfusion are discussed elsewhere in relation to the treatment of shock and haemorrhage (see Chapter 5). Major emergencies may need urgent surgery with no time for radiology as part of the primary investigation but on the other hand radiological examinations are often urgently indicated even when surgery is to be undertaken forthwith. In major multiple injuries it is often necessary to examine the whole trunk radiologically for major bony damage. In suspected renal injuries intravenous pyelography is invaluable at least to demonstrate that the other kidney is not only present but functioning. Cystograms may be indicated when there is doubt about a rupture of the urethra or bladder. The extent to which radiology will be used at the acute stage will depend on its being available without the patient having to be moved.

Some injuries arouse suspicions which should lead to further attempts to obtain information that the patient is unable to give particularly when the patient has been seen by another doctor or transferred from another hospital.

A patient arrived in a state of serious collapse on transfer from a neighbouring hospital for a primary wound repair. The collapse had occurred in the ambulance and was due to antitetanus serum received at the first hospital—fortunately noted in the accompanying letter.

The knowledge that a patient has been fully conscious after a head injury may be vital when coma has supervened from extradural bleeding.

In the more seriously injured patient, when there is time to investigate wide trunk radiographs may still be needed. Fractures of the pelvis and spine without visceral or cord damage can easily be missed at a primary examination. Later they may continue to bleed or provoke major discomfort. Radiological examination of the abdomen may show swallowed foreign bodies, gas under the diaphragm in the erect position or in the flank from an antero-posterior view taken with the patient lying on the side.

Occasionally there may be a need for stereoscopic films or tomographs and for other special techniques. The clinician and radiologist or radiographer must work closely together.

One difficulty is that some patients who appear too ill for full investigation stand most in need of accurate diagnosis. Others escape investigation simply because they seem so well and injury has appeared trivial. The latter group is likely to be neglected once preliminary investigation has revealed no obvious lesion particularly when patients with more obviously serious injuries are admitted at the same time and from the same incident. It is not always enough to admit for

observation with a half hourly pulse or blood pressure chart. Many patients with apparently minor injuries need to be re-examined at intervals in the ward. Some will appear much better than when first seen but others will show signs of more serious trouble. The rest will remain in doubt: some to recover, others to produce evidence later. Observation must continue.

REFERENCES

- Bywaters, E. G. and Beall, D. (1941). *Brit. med. J.*, 1, 432.
Fine, J., Frank, H., Schweinburg, F., Jacob, S., and Gordon, T. (1952). *Ann. N.Y. Acad. Sci.*, 55, 429.

CHAPTER 4

CIRCULATORY RESPONSES TO INJURY

I G GRABER

INTRODUCTION

Physiological adaptations to trauma

EVOLUTION has resulted in physiological adaptations to trauma which generally favour survival. The normal homeostatic mechanisms which govern circulatory and related metabolic processes and maintain the internal environment become reorientated in a manner to mitigate the effects of trauma and blood loss and to promote healing.

The changes in cardiovascular, endocrine and nervous systems are at present incompletely understood but the effects include the following.

(1) A fall in blood pressure is prevented or delayed in the face of blood loss and a diminished cardiac output by selective vasoconstriction. This redistributes the cardiac output so that the blood supply to the brain and heart is maintained at the expense of the splanchnic bed, liver, skin, muscles, and kidneys. The redistribution is mediated through the central nervous system (see Heymans, 1950; Henry, 1955; Cort, 1955) but only occurs in the presence of the adrenal cortex (Remington, 1951).

(2) A redistribution of the body water occurs so that cellular water is made available for the vascular compartment. This is mediated through the endocrine system: haemorrhage results in an increase in blood aldosterone and hydrocortisone (Farrell, Rosnagel and Raushkolb, 1956) and antidiuretic hormone (ADH) (Ginsburg and Brown, 1956) the action of which tends to reconstitute the blood volume. The alteration in the hormonal balance varies with time (Wrong, 1956) so that for some hours water is retained by the kidney (due to the action of ADH) later urine has the electrolyte composition of intracellular fluid (due to the action of aldosterone) (see Figs. 3 and 4).

(3) A loss of potassium in the urine and sweat induces hypotonia, a disinclination to movement, loss of appetite and intestinal immobility thus resting the injured part and furthering splanchnic vasoconstriction.

(4) A catabolic phase ensues (due to the action of hydrocortisone) which provides essential amino acids for tissue repair (Cutbertson, 1954) and water for the replenishment of depleted compartments.

Deleterious effects

These adaptations include the following features which militate against the reconstitution of a normal internal environment.

(1) The selective arteriolar vasoconstriction, which maintains the blood pressure in large arteries, drastically reduces the pressure and flow in the capillaries, thereby producing local hypoxia and inhibiting the exchange of fluid and metabolic products between the blood stream and the tissues. Vasoconstriction converts the skin from an organ of temperature regulation to a calorimeter cover: skin temperatures fall while

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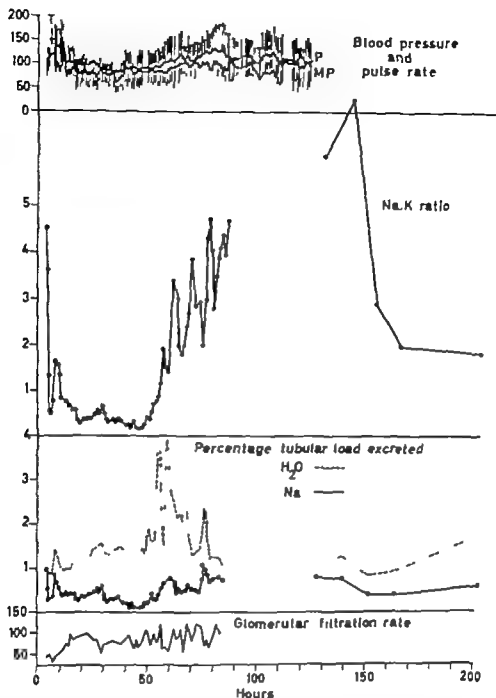


FIG. 4—Serial cardiovascular and metabolic (endocrine) effects in a patient with severe multiple injuries transfused with a large quantity of blood. T indicates induction of anaesthesia for surgery. The patterns of change in urine Na:K ratio and percentage tubular load of water and sodium excreted differ only in amplitude from those in Fig. 3. MP=mean blood pressure. P=pulse rate. Na:K ratio in the urine is a function of aldosterone excretion and the percentage tubular load of water excreted is a function of antidiuretic hormone activity.

INTRODUCTION

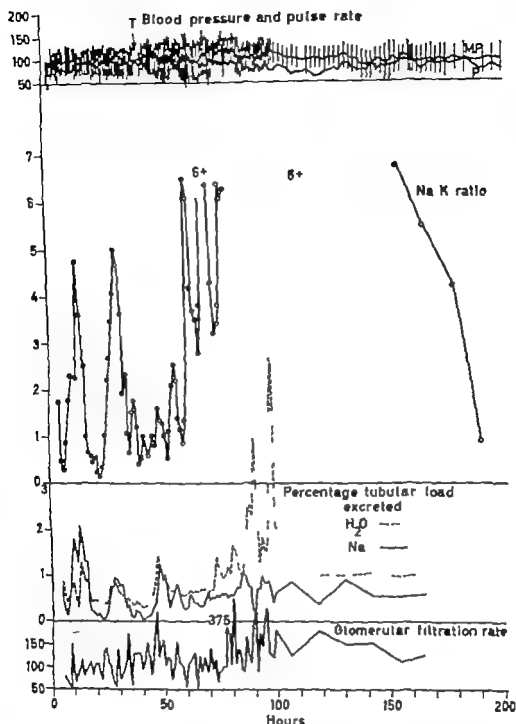


FIG 5—Serial cardiovascular and metabolic (endocrine) effects in a patient with severe multiple injuries transfused with a large quantity of blood. The pattern of change shows rapid, large fluctuations in the urine Na K ratio and the percentage tubular load of water and sodium excreted, although the blood pressure remained relatively constant and normal. MP=mean blood pressure P=pulse rate Na K ratio in the urine is a function of aldosterone excretion and the percentage tubular load of water excreted is a function of antidiuretic hormone activity

HAEMODYNAMICS OF BLOOD LOSS

Cardiac output and its distribution

The importance of a fall in cardiac output after injury and blood loss was established by Richards and Cournand and their colleagues in their studies on some 110 cases of civilian injuries. Studies of blood volume and cardiac output were made on admission and after therapy: the results (Cournand and his colleagues 1943, 1945; Lauson, Bradley and Cournand 1944; Noble and Gregerson 1946; Richards, 1943, 1948) confirmed that the reduction of blood volume was due to local blood loss into damaged tissues (Blalock 1930) and showed that the blood loss was accompanied by a decrease in cardiac output. Studies of renal blood flow measured by the clearance of para-amino-hippuric acid showed that the reduction was far greater than the fall in cardiac output: this could only occur if there was renal vasoconstriction. Simultaneous vasoconstriction of the skin was noted. Since a marked increase in the total resistance occurred it can be deduced from the data that active vasoconstriction occurred in other vascular beds besides the skin and kidneys. Active vasoconstriction in the splanchnic bed following trauma can be adduced from the data of others (Blalock and Levy 1937; Freeman, Frank and Fine 1952; Lillehei 1957) and was shown to occur in muscle by Zwelfach (1951).

A graph constructed by Wiggers (1950) showed the relationship between reduction of blood volume and cardiac output and indicated that a 33 per cent blood loss resulted in a halving of the cardiac output. Recent work indicates that the cardiac output may decrease further with time without further blood loss.

The resting cardiac output averages 5.6 l per minute in man (Brotmacher and Deutcher 1956) and is distributed to five main vascular beds: the brain, kidney, liver and splanchnic area, skin and muscle. Each of the first three receives a blood flow of approximately 1,500 ml per minute, while skin and muscle together receive about 800 ml per minute; about 100 ml per minute is supplied to the myocardium. The preferential distribution of blood to the brain and heart in the face of a reduced circulating blood volume was postulated by Starling (1909) and has been confirmed by clinical experience and experimental evidence. If the cardiac output is reduced to say 2.8 l per minute and the blood flow to brain and myocardium is maintained then 1.3 l at best are left for redistribution to the kidneys, liver, skin and muscles, which normally receive 3.8 l per minute. Ischaemic anoxia is produced and is aggravated by haemodilution and disturbances in cell metabolism (Strawitz and Hift, 1956). Lauson, Bradley and Cournand (1944) found that renal ischaemia often persisted after transfusion to normal blood volume by plasma or blood even though the cardiac output was restored.

Experimental evidence in the past 10 years has shown that neither the kidney, liver nor the intestine (Fine 1955; Lillehei 1957) can tolerate ischaemia for more than a few hours without changes in their structure and function. These contribute to a disturbance of the internal environment and may facilitate death from renal failure or possibly from circulatory collapse unresponsive to transfusion.

The ability to reconstitute a normal blood volume following trauma is limited: an acute blood loss of 4.5 per cent of the body weight is almost invariably fatal (Wiggers 1950). In man this represents 4 per cent of the total body water, 13 per cent of the available sodium, 0.5 per cent of the available potassium and 50 per cent of the circulating red blood cells. The haemodilution following blood loss

which persists for weeks as the anaemia of trauma (Topley and Clarke 1956) reveals man's limitation in replacing erythrocytes. Early haemodilution is not accompanied by a significant fall in plasma proteins (Gregerson and Root 1947, Beecher 1949) indicating that the body has significant reserves of protein. The reserves of body water are also considerable and 4.2 l. of water (6 per cent of body weight) must be lost before clinical manifestations of dehydration occur. Therefore the major need for replacement therapy after trauma and blood loss is red blood cells and these are best given in the form of whole blood.

Quantitative assessment of blood loss and its replacement

Perhaps the most significant recent advances in treatment are first, the recognition of the large amounts of blood necessary to restore and maintain a normal blood volume following severe multiple injuries and, secondly the elimination of plasma and plasma substitutes as forms of therapy.

Summarizing the work of his group in 1948 Richards stated: 'It needs no emphasis at this time to record the extraordinary recovery that can take place even in advanced shock by abundant replacement with plasma and whole blood. In others there will be temporary resuscitation followed by gradual failure and, again death. Shock is very much more than a disturbance in blood flow. Richards' concept of abundant replacement was based on the work of Noble and Gregerson (1946) who concluded that in severe trauma on the average 1.5 to 2.0 litres of blood are required for replacement. This is often a gross underestimation. Volume measurements of injured limbs supplemented by blood volume studies have shown that 2 l. of blood or more can be lost into injured muscle after a simple fracture of the femur or extravasated subcutaneously following a fractured pelvis (Grant and Reeve, 1951; Clarke, 1952; Clarke, Topley and Flear 1955). This is in accord with the findings of Duncan and Blalock (1942) who estimated the extravasation of blood into traumatized limbs of animals by weighing.

The experience of Clarke and his colleagues (1959) in civilian trauma and Prentice and his colleagues (1954) and Artz and his colleagues (1955) in war injuries, is that transfusion of as much as 10-25 l. of whole blood may be necessary to maintain the blood volume near normal after severe injuries. Much of this is needed to replace bleeding into the tissues during and following resuscitation as well as the blood lost during surgery and in the post-operative period.

Blood loss at any time during treatment must be adequately replaced so as to avoid or reduce the deleterious effects of severe hepatic and particularly renal ischaemia. Animal experiments suggest that bacterial toxins from ischaemic intestine may be important in prognosis since they may cause widespread vasodilatation and profound hypotension not influenced by blood transfusion or vasoconstrictor drugs. Arterial perfusion of the splenic vein (Delorme, 1951) and splanchnic bed (Lillehei 1957) and antibiotics (Fine, 1955) can substantially reduce the mortality of prolonged splanchnic ischaemia. This work supports the hypothesis of earlier workers that a toxic factor produces irreversible hypotension following trauma (for further references see Wiggers 1950) but emphasizes that ischaemia is the pathogenetic mechanism. Nevertheless there is no definite evidence that toxic or bacterial factors play an important part in man except in the special cases of fulminating gas gangrene or early peritonitis.

HAEMODYNAMIC EFFECTS OF THERAPY

The time taken for a normal cardiac output and blood distribution to occur once a normal blood volume is established is determined by many factors. These include the time between injury and transfusion, the rate of transfusion, the amount of colloid given before blood transfusion begins, the salt and water losses, whether vasoconstrictor drugs have been used, whether general anaesthesia and surgery are employed as well as the nature and situation of the injuries.

Colloid therapy

The arterial oxygen content may fall considerably after trauma because of haemodilution. Thus Cournand and his colleagues (1943) found a reduction to as low as 6.6 volumes from the normal value of 16.6 volumes of oxygen per 100 ml. Administration of colloids such as plasma or dextran further reduces the oxygen-carrying capacity of the blood (Beecher, 1949). By increasing the plasma volume, colloids reduce the space available for transfusion of whole blood and make rapid reconstitution of the oxygen-carrying capacity of the blood impossible. This may even decide a fatal issue (Parkins, Permutt and Vars, 1953).

Citrate effect of blood transfusion

Stored blood is generally diluted about 1 in 4.5 with 3.8 per cent solution of sodium citrate. Hejhal and Firt (1954) and Firt and Hejhal (1957) demonstrated that intravenous 3.8 per cent solution of sodium citrate, in doses equivalent to 100 ml. in man, causes a rise in pulmonary artery and right atrial pressure in the dog. After haemorrhage, the transfusion of citrated blood, at rates equivalent to 420 ml. per minute in man, caused the central venous and pulmonary arterial pressures to rise sharply and the systemic blood pressure to fall. Signs of over-transfusion appeared before the blood volume had been replaced. Heparinized blood had no such deleterious effects. These authors gave case details of similar effects in man during ultra-rapid transfusion of citrated blood; they suggested that intravenous calcium gluconate and 1 per cent procaine should be given. Ludbrooke and Wynn (1958) pointed out that a straight line relationship exists between the rate of blood transfusion and the plasma citrate concentration. They estimated that a rate of 180 ml. per minute was the upper limit of safety in normal man and predicted that citrate intoxication could occur at much slower rates of transfusion if liver impairment was present, since its disappearance rate in man is dependent on liver function. They also urged calcium therapy.

Water and electrolytes

The necessity for adequate replacement of serious salt and water losses hardly needs to be emphasized, since pure sodium depletion may produce a clinical picture and metabolic response very similar to that of haemorrhage: the cardiac output falls rapidly, perhaps to 40 per cent of normal, followed by a prolongation of circulation time and an increase in total resistance. These effects are intensified if water losses occur (Elkington, Danhowski and Winkler, 1946).

The basis of replacement must for the present rest upon clinical assessment together with accurate measurement of losses from the gastro-intestinal tract and

HAEMODYNAMIC EFFECTS OF THERAPY

urine rather than on serum levels. Following severe trauma the serum sodium level often falls to 120–125 mEq per litre and is accompanied by the retention of sodium by the kidney. The total body sodium is unchanged but the redistribution is largely mediated by aldosterone which promotes the migration of sodium into the cells (Howard, Prawley and Artz, 1955). These low sodium levels produce no obvious clinical manifestations and are relatively resistant to change by sodium therapy.

Sodium replacement by normal saline solution has two disadvantages: first, it is acid relative to blood and secondly it increases the renal loss of potassium. The hypersecretion of aldosterone causes increased concentrations of potassium in the urine. Sweat and intestinal secretions. An amount of potassium equivalent to that in the whole extracellular space may be lost in 500 ml. of urine and as much as 400–500 mEq may be lost in the first few days after severe injury. In most cases the great loss of potassium is relatively short lived and is apparently insufficient to produce significant clinical effect, but if the loss is prolonged or promoted by injudicious therapy or enhanced by specific injuries, hypokalaemia may occur. Hypokalaemia can affect unstriated muscle and cardiac muscle in movement, decreased cardiac output and even respiratory and cardiac failure. Various cardiac arrhythmias may appear and electrocardiographic changes include T wave depression, S-T depression and the appearance of U waves (Saurawicz and Lepeschikine, 1953). The clinical and physiological manifestations are more dependent upon the rate of change of serum level relative to the cellular potassium content than upon the absolute value of either. Furthermore, acutely oligoemic animals are 10 times more sensitive to rapid changes in serum potassium than are normal animals. The replacement of serious potassium losses is best undertaken after the blood volume has been returned to normal and at rates not exceeding 20 mEq per hour because of the possibility of hyperkalaemia with its cardiac risk. The first clinical manifestation of hyperkalaemia is cardiac arrest.

Hyperkalaemia is an accompaniment of oliguric renal failure (see Chapter 7).

Vasoconstrictor drugs

The use of vasoconstrictor drugs such as noradrenaline to maintain blood pressure in the face of a low blood volume can be exceedingly dangerous. Noradrenaline decreases the blood flow to the kidney, liver and splanchnic area and increases the total resistance. Deleterious processes due to ischaemia, particularly renal failure, may be precipitated.

Anaesthesia

General anaesthesia dilates the vessels in skin and muscle and increases their blood flow 4-fold (Abramson, Grollman and Schwartz, 1941; Lynn and Shackman, 1951; Shackman and Graber, 1952). The peripheral vasodilatation often diverts as much as 2–4 l of blood from the central organs. Cyclopropane decreases the cardiac output and increases the peripheral resistance through splanchnic and hepatic vasoconstriction. The combination of prolonged anaesthesia surgery and blood loss reduces the skin and muscle blood flow simultaneously with a further fall in cardiac output, an increase in total resistance and a further decrease in liver blood flow (Shackman, Graber and Melrose, 1952, 1953; de Wardener, 1953).

The increase in skin and muscle blood flow is a factor in the circulatory collapse which may follow induction of anaesthesia in oligaemic patients. Transfusion should begin before anaesthesia is induced and if possible the blood volume should be restored to normal before surgery is begun. Sudden cessation of oozing during operation is an indication for further transfusion. In these patients anaesthesia should be terminated as soon as possible because prolonged splanchnic ischaemia may be related to untreatable circulatory collapse.

SPECIFIC INJURIES

At least three types of injuries prevent the reconstitution of a normal circulatory pattern by blood transfusion alone.

Intestinal Injuries

Studies by various workers have shown that abdominal and particularly intestinal injuries are associated with considerably higher haematocrit values than skeletal injuries. This indicates that body water normally available for reconstitution of the blood volume is diverted into the intestine or the peritoneal cavity. The coils of oedematous intestine found at laparotomy in intestinal injuries probably account for most of this fluid diversion initially. Hormone secretion is not responsible since ACTH, adrenaline and ADH all decrease intestinal secretions. When the normal turnover and exchange in the gastro-intestinal tract of 8-9 L per day is considered, the great fluid losses which may occur from paralytic ileus, intestinal fistulae and aspirations are easily understood. Added to these may be losses from peritonitis. Sodium and potassium losses may also be considerable. Normally the secretions into the gastro-intestinal tract contain 10 mEq per l or less of potassium but the content may rise to 70 mEq per l when fluid loss is copious. The sodium content of intestinal losses varies with the level from which the fluid is lost. Sodium, potassium, and fluid losses can seriously reduce cardiac output so that replacement therapy is urgent and must be based on analyses and records of the amounts lost on the case history and on the hormonal state of the patient (Graber, Beaconsfield and Daniel, 1956).

Head Injuries

Severe head injuries are often associated with other injuries demanding urgent transfusion. Hesitancy in restoring the blood volume to normal owing to fear of causing intracranial haemorrhage is unjustified, since the brain receives its normal blood flow in any case. There may be a greater risk of increased cerebral bleeding if transfusion is withheld because many patients react to trauma and blood loss by hypertension since cerebral vasoconstrictor fibres do not respond to blood loss (Heymans and Bouckaert, 1933, 1935) the brain is then likely to receive more than its normal blood supply. Adequate transfusion returns the blood pressure to normal. Moreover, delaying transfusion promotes hypoxia with adverse effects on cerebral function.

Certain patients, particularly those with brain-stem lesions, develop persistent hyperventilation which results in excessive losses of carbon dioxide and water. Prolonged apnoea causes a fall in cardiac output, cerebral vasoconstriction and

SPECIFIC INJURIES

peripheral venous stasis. Tracheotomy required to prevent lung complications reduces the dead space and increases the carbon dioxide and water losses. In some cases hypernatraemia, hyperchloraemia and acidaemia develop and cause further circulatory disturbances and perpetuate the hyperventilation. Preliminary studies suggest that inhalation of 5 per cent carbon dioxide in oxygen may lower the respiratory rate and help to correct the electrolyte imbalance in some cases. Sodium and water losses in the urine if excessive, may adversely affect the circulation. It is important to measure the urinary output of patients unconscious with head injuries because replacement may be needed to prevent both dehydration and sodium depletion.

Thoracic injuries

Thoracic injuries can reduce the cardiac output out of all proportion to blood loss or even in the presence of a normal blood volume. The heart is a double pump—a right, or low pressure system and a left, or high pressure system—the outputs of which are exactly co-ordinated. These are separated by the pulmonary vascular bed, which normally contains some 500–1 000 ml of blood. A discrepancy in output of 5 per cent between the two pumps would flood the lung field with 2.7 l. of blood in 10 minutes. This may be important in chest injuries since clinical observations have shown the liability for pulmonary congestion and oedema to develop during transfusion before blood loss has been replaced.

The large vessels entering the heart, the atria and pulmonary vessels are well supplied with baroreceptors (Aviado and Schmidt, 1955) which are very sensitive to pressure change (Schaefer 1951) and may provide a mechanism for adjustment of output between the right and left sides of the heart. Reflexes initiated by these receptors produce alterations in venous return brought about by variations in posture (Henry 1955) or moderate haemorrhage (Henry Gauer and Sieker 1956).

Experiments have shown that raising the left atrial pressure by inflation of a balloon inserted into the left atrium sharply increases the pulmonary artery pressure (Henry Gauer and Reeves 1956; Henry and Pearce, 1956). Although the cardiac output falls, the urine output rises considerably and its sodium content is increased. The blood pressure is unchanged. The inflated balloon in the left atrium simulates an increased venous return to the left heart. Reflexes designed to correct this including pulmonary vasoconstriction and diuresis occur even though there is no increase in pulmonary blood flow or peripheral blood volume. These findings have relevance to chest injuries because the changes of pressure induced in these experiments are easily attained in thoracic injuries.

Crush injuries to the chest wall tension pneumothorax and haemothorax all increase the intrathoracic pressure and may reduce the venous return to the right atrium by reducing the pressure gradient between the thoracic and peripheral veins. At the same time the output of the right heart may be reduced through compression of the pulmonary vascular bed. laceration of lung tissue local haemorrhage and arterial spasm. The reduction of alveolar surface promotes anoxia and carbon dioxide retention. The former produces pulmonary hypertension by vasoconstriction (Motley and his colleagues 1947) and the latter peripheral

vasoconstriction. The rise in intrapulmonary pressure may also precipitate peripheral venous spasm and a fall of cardiac output (Braunwald and his colleagues, 1957).

Should a section of the chest wall lose its rigidity owing to multiple fractures of ribs, paradoxical breathing may occur. This has special risks (see Chapter 15). On inspiration the lung on the affected side collapses and pumps air rich in carbon dioxide into the expanding opposite lung; on expiration it expands and sucks in expired air containing additional carbon dioxide from the contracting healthy lung. The partial pressure of carbon dioxide (P_{CO_2}) is thus raised, increasing the blood P_{CO_2} . The initial effects of this are vasoconstriction in muscle, skin and the splanchnic bed and an increase in cerebral blood flow. Further increases cause vasomotor paralysis and heart block.

An attempt to reconstitute a normal cardiac output and distribution of blood in patients with severe chest injuries may fail unless simultaneous steps are taken to return the intrathoracic pressure to normal to re-expand the lung fields and combat paradoxical breathing.

LIMITATIONS OF CLINICAL SIGNS AS A GUIDE TO BLOOD LOSS AND TRANSFUSION

The timing of blood transfusion becomes an urgent clinical problem when transfusions amounting to 4 or even 6 times the blood volume are involved, since overtransfusion is a possible risk. Although records from the Korean War contain only 3 cases of possible overtransfusion and its occurrence is rare at the Birmingham Accident Hospital, Downs (1958) expressed a common but erroneous view when he stated "circulatory overloading is now probably the most common cause of death" in treatment of massive blood loss. His data indicated that barely adequate quantities of blood were transfused too late and too fast. Downs' paper however is a pointed reminder that the clinical problems raised by Paget (1862) remain unsolved. Paget recognized that clinical signs were not necessarily related to the severity of the injury nor was prognosis dependent on initial symptoms.

In studies of civilian and battle trauma efforts have been made to correlate clinical symptoms and signs to the degree of blood loss but the findings are contradictory. Thus, Fisher (1958) agreed with Grant and Reeve (1951) that there is a possible correlation between pulse rate and blood loss, while Evans and his colleagues (1944) and Beecher (1949) could find no such correlation. Keith (1919), Evans, Beecher and Grant and Reeve all found a correlation between a fall in systolic blood pressure and oligæmia, although they disagreed widely as to the extent of the fall. Fisher however found the blood pressure of less value in diagnosis of blood loss. Furthermore, the graphs relating red-cell volume, plasma volume or blood volume on the one hand, to blood pressure, pulse rate, skin temperature and venous tone on the other are scattergrams from which trends may be deduced and statistical correlations assessed; however they are of limited value in assessing the blood lost in an individual patient.

The failure to obtain reasonable correlations indicates great complexity of mechanisms and limitations of certain techniques. In general three main groups of factors are involved: (1) an alteration of the circulation and metabolism with time which has been referred to earlier; (2) changes in the meaning of physical

CHANGES IN THE MEANING OF MEASURES AND PHYSICAL SIGNS

signs and measurements after trauma and (3) the nature of the disturbance in self regulating mechanisms—these require consideration

CHANGES IN THE MEANING OF MEASURES AND PHYSICAL SIGNS

The changes in blood pressure, pulse rate, central venous pressure and peripheral blood values have haemodynamic consequences not linearly related to changes in blood volume

Blood pressure

The relationship between blood pressure and cardiac output may conveniently be considered to follow a simplified form of Poiseuille's law blood pressure = cardiac output \times total resistance. The overall or total resistance (R) is a complex made up of several functions one of which is that flow in a tube is proportional to the fourth power of its diameter. Thus, small changes in the diameter of arterioles can easily shunt blood from one vascular bed to another. The main vascular beds of the body—kidney, splanchnic and liver, skin and muscle, and brain—exist in parallel. Thus in the systemic circulation—

$$\frac{1}{R} \text{ (total)} = \frac{1}{r} \text{ kidney} + \frac{1}{r} \text{ splanchnic} + \frac{1}{r} \text{ muscle and skin} + \frac{1}{r} \text{ brain}$$

The total resistance is thus less than the resistance in any single component, so that a large increase in a regional resistance (r) would not necessarily affect the total resistance indeed it may be associated with a fall in the total resistance if that in other regions is simultaneously reduced. This explains why a normal blood pressure and cardiac output (CO) can occur in the face of marked renal or hepatic ischaemia.

In considering haemodynamic states three possible clinical categories have to be borne in mind (1) low blood pressure = normal or high CO \times low R, low CO \times low R, or very low CO \times high R. (2) normal blood pressure = normal CO \times normal R, high CO \times low R, or low CO \times high R. (3) high blood pressure = high CO \times normal R, normal CO \times high R or low CO \times very high R. Viewed in this way it can easily be seen that blood pressure *per se* can be a source of false comfort or unnecessary anxiety. This does not of course underestimate the clinical importance of sustained hypotension as an adverse sign following severe injury or blood loss.

Pulse wave and pulse rate

Pulse rate in equilibrium states is proportional to the cardiac output but this relationship does not hold after trauma and blood loss. Alexander and Webb (1947) found that the mean pressure in the dorsalis pedis artery was higher than that in the femoral artery and aorta in haemorrhage-induced hypertension and this was confirmed in anaesthetized normotensive dogs (Graher and Beaconsfield, 1955). Studies of alteration in the pulse wave in the three arteries led Alexander and Webb to conclude that the higher pressure in the smaller arteries was due to a summation effect caused by the elasticity of the arterial wall. Garton (1958) re-examined this data and concluded that the higher pressure means that active work is done by the walls of the smaller arteries. This would alter pressure flow

relationships in the presence of vasoconstriction and thus also alter the meaning of pulse rate and blood pressure

Blood-volume estimations

Although blood volume estimations have been of great help in demonstrating the importance of oligæmia their value is limited in the early phases of trauma. The validity of the estimation rests on three assumptions that the substance injected remains in the vascular tree that it becomes uniformly mixed and that it can be accurately estimated.

The value of the vital red and Evans blue (T 1824) techniques is limited by loss of dye from the blood stream and the difficulty in their estimation in the presence of haemolysis and lipaemia. The use of erythrocytes labelled with ^{51}Cr and ^{32}P overcomes these difficulties but not those of mixing. In normal patients the mixing time for erythrocytes and Evans blue is 3-5 minutes (Lauson and his colleagues 1947). After severe haemorrhage or retransfusion it may be delayed or unpredictable. Delorme, Mukherjee and Rowlands (1952) found that in dogs the mixing was delayed for 3-5 hours after haemorrhage and after retransfusion. This was also the experience of Prentice and his colleagues (1954). Our own studies have shown that mixing time may be disturbed for even 10 days after severe injury. Fig. 6 illustrates mixing irregularities which occurred in a severely injured patient. The basis of these irregularities lies in the complexity of the capillary circulation after trauma (Zweifach and his colleagues 1944, Zweifach 1951) and regional alterations in vascular tone.

A blood volume estimation on admission can give no indication of how much bleeding will occur after transfusion; this can only be assessed clinically. Repeated red cell volume estimations can be of value as a guide to the adequacy of transfusion (Clarke and his colleagues 1959).

Even if the blood volume estimate is accurate its interpretation as a guide to transfusion may be difficult because of the variation in normal volume among different people: errors in transfusion can be made. The error can be reduced if lean body mass instead of body weight is used as a criterion of normal blood volume (Muldowney 1957). Other errors are due to ignoring the body venous haematocrit (Stead and Ebert, 1941) and failure to assess continuing bleeding.

Central venous pressure

The central venous pressure is a resultant of the blood volume, capacity of the veins, venous return, cardiac output and intrathoracic pressure, the last named depending upon the volume of the thoracic cage and intrathoracic blood volume. Changes in any one of these may be reflected in a change of pressure.

Withdrawal of up to 15 per cent of the blood volume leads to a small temporary fall in right atrial pressure. However, after withdrawal of 20 per cent or more or if there is pain or emotional disturbance peripheral venospasm occurs and upsets the pressure-volume relationships (Page and his colleagues, 1955). Venospasm also develops when there is a fall of systemic blood pressure and raises the venous pressure. Thus, Wiggers (1950) recorded animals dying of haemorrhage with central venous pressures higher than normal.

CHANGES IN THE MEANING OF MEASURES AND PHYSICAL SIGNS

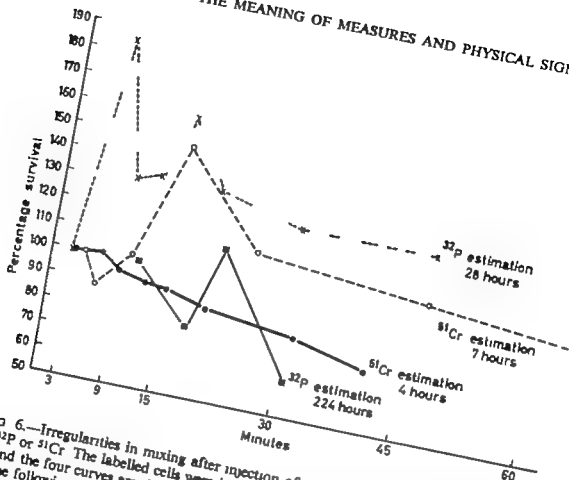


FIG. 6.—Irregularities in mixing after injection of red cells labelled radioactively with ^{32}P or ^{51}Cr . The labelled cells were injected at 4, 7, 28 and 224 hours after injury and the four curves are the respective serial values obtained from the blood during the following 30–60 minutes. The level of radioactivity in the blood at 3 minutes after injection was taken as 100 per cent survival. Note that errors of over 50 per cent in blood-volume estimation can easily be made if many of the points are taken to represent true mixing. The labelling and counting of red blood cells was carried out by Dr J. Davies. Data was derived from the same patient as Fig. 4.

Peripheral blood values

The body water is distributed in three compartments—vascular, interstitial and intracellular. The interstitial and intracellular compartments consist of discrete subdivisions such as the skin, muscle, liver, intestine, kidney and lungs which are concerned with specific functions in controlling the internal environment. Each local function sets up a local difference which is transmitted to the vascular compartment. The blood stream is thus the integrator of the internal environment.

Water exchange between the interstitial and intracellular spaces occurs across cell membranes which maintain a large differential in ionic composition and osmolality between the two spaces (see Table) by mechanisms which are hormone dependent. Aldosterone promotes a loss of potassium from cells and a rise in cellular sodium and acid whilst hydrocortisone causes a shift of water and electrolytes out of cells and antidiuretic hormone probably causes an increase in cellular water.

TABLE

COMPARISON OF THE CATION AND ANION CONCENTRATIONS IN EXTRACELLULAR FLUID AND WITHIN CELLS

Extracellular fluid cations		Cell				Extracellular fluid anions	
		Cations		Anions			
Na ⁺	137	K ⁺	150	HPO ₄	100	Cl	106
K ⁺	5	Mg ⁺⁺	40	Protein	66	HCO ₃	30
Ca ⁺⁺	3	Na	5	SO ₄	18	Organic acids	8
Mg ⁺⁺	1			HCO ₃	11	HPO ₄	2
						SO ₄	1
						Protein	1
Total	146		195		195		146

Numbers are in milliequivalents.
 Note that the Na : K ratios in the extracellular and cellular fluids are almost reciprocal. Urine after trauma often has Na : K ratios similar to those in cellular fluid.

These considerations are important in trauma since the vascular beds which undergo vasoconstriction suffer a gross disturbance of fluid exchange with the interstitial space. The narrowing of the arterioles causes a steep fall of pressure across their length whilst the volume of flow falls proportional to the fourth power of the diameter. As the capillary pressure falls water tends to be retained inside the capillaries through the relatively unopposed osmotic pressure of the plasma proteins. The slow flow permits an increased escape of protein and thereby impairs the flow of interstitial water into the blood stream. Products of cellular metabolism accumulate locally. Changes in hormonal balance occur which alter the ionic composition of intracellular and interstitial fluid. In this way peripheral venous samples become only obliquely representative of local metabolism while mixing in the general circulation is disturbed by altered regional flow rates. Thus the mixing time for tritium is increased by several hours after trauma (Howard and Scott, 1955).

The altered circulation in the kidney, skin and gastro-intestinal tract interferes with water and ionic exchange and prevents adjustment to losses.

DISTURBANCE OF SELF REGULATING MECHANISMS

When the physiological changes which follow trauma with blood loss and the subsequent clinical intervention are recorded at frequent intervals, they show features of a kind to be expected in the operation of a complex system of automatic and interacting regulatory mechanisms (Figs. 3-5).

The composition of the blood, its circulation and its distribution are normally maintained within close limits by regulating mechanisms. Each quantity regulated involves an element which plays the part of a responsive measuring instrument. Through this detector and through mechanisms that include nervous, humoral and vascular elements, the tendencies to depart from normal values are corrected.

DISTURBANCE OF SELF REGULATING MECHANISMS

Self limitation of variation by "feed back" from a measuring element is referred to as feed back control

It is well known to engineers that when regulating systems depending on feed back are subjected to disturbance they may pass through a period of oscillation before returning to the steady state. Certain forms of interference can modify this reaction so that the whole system may be maintained indefinitely in a state of oscillation (Tustin, 1957 1959)

Similar fluctuations occur in body physiology after trauma. The interrelationships are not linear but if they were known quantitatively and in detail the behaviour of the system might be predicted and therapy might be designed to avoid the extreme fluctuations and conditions which may result in irreversible damage. For the present this is not possible but the recognition of this situation is relevant to therapy in its implication that little significance should be attached to single determinations of certain physical signs in the early period after trauma.

Studies have been made of serial changes in blood pressure, pulse rate, glomerular filtration rate, aldosterone excretion (as measured by the sodium-potassium ratio in the urine) and secretion of antidiuretic hormone (by calculating the percentage of filtered water or tubular load excreted) in response to trauma and the alterations produced by blood transfusion, anaesthesia and surgery. Measurements were made very frequently during the first few days. When the relationships were plotted they resembled those which one would expect if a series of feed back control mechanisms were seriously unbalanced (Figs 3-5)

Figs 3 4 and 5 are compared the last two were derived from previously healthy young men who suffered multiple injuries and were transfused with more than 10 l. of blood in the first 24 hours whilst the data in Fig. 3 came from a patient with a fractured spine and tibia who was not transfused. Figs. 3 and 4 show patterns of endocrine response (urine sodium-potassium ratio and percentage excretion of tubular water load) which are very similar except for a later increase in amplitude in Fig. 4 whilst the blood pressure and pulse rate show wide periodic fluctuations in Fig. 4. On the other hand Fig. 5 shows rapid and large fluctuations in endocrine response with a constant blood pressure. These individual differences in the response to trauma and therapy make it difficult to assess the blood volume from physical signs.

Serial estimations of cardiac output in the early phases of trauma have not yet been reported, mainly because of technical difficulties. Recent advances in technique such as determinations employing ^{132}I (Veall and his colleagues 1954 Veall and Vetter 1958) and blood conductivity (Goodwin and Sapirstein, 1957) are suitable for repeated estimations and interfere minimally with the patient. Investigations of changes in cardiac output relative to the circulatory and metabolic patterns are at present under investigation and it is possible that the results may clarify some of the problems of therapy.

REFERENCES

- Abramson, D. L., Groffman, A. T. and Schwartz, A. L. (1941). *Anesthesiology* 2, 186.
Alexander, R. S., and Webb, E. A. (1947). *Amer J Physiol.*, 150 272.
Ariz, C. P. Howard J. M. Sako Y., Bromwell A. W., and Premice T. (1955) *Ann Surg.*, 141 285.
Aviado D. M. Jr., and Schmidt, C. F. (1955). *Physiol Rev* 35 247.

- Beecher H K. (1949) *Resuscitation and Anesthesia for Wounded Men the Management of Traumatic Shock* Springfield Thomas.
- Blalock, A. (1930). *Arch. Surg.*, 20 759
- and Levy S. E. (1937) *Amer J Physiol.*, 118 734
- Brauwald, E. Binion J T., Morgan W L., Jr., and Sarnoff S J (1957) *Circulat Res.* 5 670.
- Brotmacher L. and Deutcher D C. (1956) *Clin. Sci.* 15 441
- Clarke, R. (1952). *Ann. R. Coll Surg Engl.*, 2, 87
- Topley E. and Flear C. T G (1955) *Lancet* 1 629
- — Davies, J W L. and Fisher Mary R. (1959) In the press.
- Cort, J H (1955) *Physiol bohém.* 4 14
- Cook, R. E. Segar W E. Cheek, D B., Colville F E. and Darrow D C. (1952) *J clin. Invest* 31 798
- Courmand, A., Riley R. L. Breed, E S Baldwin E F and Richards, D W Jar (1945) *J clin. Invest* 24 106.
- — Bradley S E. Breed, E S Noble, R. P. Lauson, H D., Gregerson, M I and Richards, W D (1943) *Surgery* 13 964
- Cuthbertson, D P (1954) *Brit med. Bull* 10 33
- Delorme E. J (1951) *Lancet* 1 259
- Mukherjee S. R. and Rowlands, S (1952) *Quart J exp Physiol.*, 37 107
- Doniach, I (1958) Personal communication.
- Downs, J W (1958) *Ann Surg* 148 73
- Duncan, G W and Blalock, A. (1942) *Ann Surg.*, 115 684
- Elkington J R. Danhowski T S. and Winkler A W (1946) *J clin. Invest* 25 120
- Evans, E. I Hoover M. J Watson Jones, G III and Alma T (1944) *Ann Surg* 119 64
- Farrell C. L. Rosnaple, R. S. and Raukolb E. W (1956) *Circulat Res* 4 606.
- Fine J (1955) *Ann Surg.*, 142 361
- Firt, P and Hejhal L. (1957). *Lancet* 2, 1132.
- Fisher Mary R. (1958) *Clin Sci.*, 17 181
- Freeman E. W Frank H A. and Fine J (1952) *Ann. Surg* 134 70
- Garton, C. G (1958). Personal communication.
- Ginsburg, M. and Brown, L. M (1956) *Brit J Pharm. Chemother* 11 236.
- Goodwin R. S. and Sapirstein, L. A. (1957) *Circulat Res* 5 531
- Graber I G. and Beaconsfield, P (1955). Unpublished data.
- — and Daniel O (1956) *Brit Med J.*, 1 778
- Grant, T and Reeve, E. B (1951) *Spec Rep Ser med. Res. Comm. Lond.*, No. 277 London H M Stationery Office.
- Gregerson M I and Root W S. (1947) *Amer J Physiol* 148 98
- Hejhal L. and Firt, F (1954) *Oldsky Lécent Prudkého Krdcentl. Prague* Nakladatelství Československé Akademie Ved.
- Henry J P (1955) *W.A D C Tech. Rep.*, 55 478
- and Pearce, J W (1956) *J Physiol* 131 572.
- Gauer O H. and Reeves, J L. (1956). *Circulat Res.*, 4 85
- — and Sieker H O (1956). *Ibid.* 4 91
- Heymans, C. (1950) *Introduction to the Regulation of Blood Pressure and Heart Rate* Springfield Thomas.
- and Bouckaert, J J (1933) *J Physiol* 79 49
- — (1935) *Ibid.*, 84 367
- Howard J M Prawley J P and Artz, C. F (1955) *Arch. Surg Chicago* 71 205
- and Scott, R., Jr (1954) *Surg Gynec Obstet* 199 707
- Keith, N M (1919). *Spec. Rep Ser med. Res Comm. Lond.* No 27
- Lauson, H D Bradley S. E. and Courmand A. (1944). *J clin Invest* 23 381
- Overbey D T., Moore, J C and Shadle O W (1947) *Amer J Physiol.*, 151 282, 290 297 303
- Lillehei R. C. (1957) *Surgery* 42, 1043
- Ludbrooke, J., and Wynn V (1958). *Brit med. J* 2 523
- Lynn, R. II and Shackman, R. (1951). *Brit med. J.*, 2, 333
- Macpherson, C. R. (1956). *Brit J exp Path.* 37 279
- Merriman, J E. (1954) *Proc. 4th Conference Josiah Macy Foundation* p 208

REFERENCES

- Motley H. L., Courmand A Werk O L., Himmelstein, A and Dresdale, D (1947) *Amer J Physiol.*, 150 315
- Mukdowney F P (1957). *Clin Sci.*, 16, 163
- Noble R. P and Gregerson, M I (1946). *J clin. Invest.*, 25 158 172.
- Page E. B., Hickman, J B. Seiker H O McIntosh, H D and Pryor W W (1955). *Circulation* 11 262.
- Paget, J (1862) *Brit med J* 2, 157
- Parkins, W M., Permutt, J H., and Vars, H M (1953) *Amer J Physiol.*, 173 403
- Prentice, T C Olney J M., Jnr., Artz, C P and Howard J M (1954). *Surg Gynec Obstet.*, 99 542.
- Remington, J W (1951). *Amer J Physiol.*, 165 306.
- Richards, D W Jnr (1943). *Harvey Lect.*, 217
- (1948). *Ann. N Y Acad Sci* 49 534
- Saurawicz, B., and Lepeschkin E. (1953). *Circulation* 8 801
- Schaefer H (1951) *Ergebn Physiol.* 46 71
- Shackman, R., and Graber I G (1952) *Brit J Surg.*, 161 13
- — — (1953). *Clin Sci.*, 12, 307
- Short E. Zweifach B. W., Furchgott R. F., and Baez, S (1951) *Circulation* 3 42.
- Stanbury S W (1957). *Advan Intern Med.* 9
- Stirling, E. H. (1909) *The Fluids of the Body* London Constable.
- Stead, E. A., and Ebert, R. V (1941) *Amer J Physiol* 132, 411
- Strawitz, J C and Hift, H (1956). *Proc. Soc exp Biol NY.*, 91 641
- Topley E., and Clarke R. (1956). *Blood* 11 357
- Tustin A. (1957). *The Mechanism of Economic Systems* London Heinemann.
- (1959) Personal communication.
- Veall, N., and Vetter R. (1958). *Radioisotope Techniques In Clinical Research and Diagnosis* London Butterworth.
- Pearson, J D., Hankley T H., and Lowe, A. E. (1954). *Radioisotope Conf.*, 1 183
- de Wardener H. E. (1953). *Clin Sci.*, 12, 169 175
- Wiggers, C. J (1950) *Physiological Shock* p 42. New York The Commonwealth Fund.
- Wright, R. D., and Devine J (1944) *Med. J Aust* 1 21
- Wrong, O (1956) *Clin. Sci* 15 403
- Zweifach, B W (1951). *Trans Int Conf on Shock* New York Josiah Macy Jnr Foundation.
- Lee, R. E. Hyman, C. and Chambers, R. (1944) *Ann. Surg.*, 120 232.

CHAPTER 5

RESUSCITATION AND TRANSFUSION IN SEVERE INJURIES

RUSCOE CLARKE

HAEMORRHAGE is the most important single factor in traumatic shock and whole blood transfusion the most important recent advance in therapy. The diagnosis and treatment of the injured patient should be approached with the object of establishing not only the nature and extent of injury but also the amount of blood lost and its relationship to the clinical and circulatory state. Transfusion and other therapy can then be based on a dynamic picture of anatomical injury and physiological changes with the aim of preventing pathological and undesirable sequelae.

Grant and Reeve (1951) discouraged the use of the word "shock" because of its clinical vagueness. Pickering (1958) used the word "shock" to illustrate how a pseudo-scientific label can hold up progress. He suggested that Cannon and Baylis went wrong in their support for the toxic theory of shock because they assumed that the word represented a clinical entity—"that because the same word was used, therefore the circumstances leading to the final picture were the same". The point is well made but his explanation is not quite accurate. Cannon (1919) was certainly familiar with clinical realities during World War I. The one thing missing from his experience was that he had never seen the effects of adequate transfusion. This only became possible with the advent of stored blood so that it was difficult at one time to judge between the conflicting views of Cannon (1923) and Blalock (1940). The test of practice was carried out during the Spanish Civil War, World War II, and later during the Korean War. The relevance of war experience to civilian trauma has been demonstrated by workers at the Birmingham Accident Hospital. The contribution of blood loss has been studied quantitatively as well as qualitatively and as a result, we can begin to evaluate some of the other factors that influence the clinical state and prognosis.

Signs of systemic upset

Grant and Reeve (1951) described patterns of early circulatory response to injury and their relation to blood loss and oligæmia assessed from blood-volume studies. The patterns have been re-assessed for civilian injuries by Fisher (1958) who simplified their number and related them to red-cell volume as well as total blood volume measurements. Fisher considered the clinical states at the actual time of blood-volume measurement and thereby avoided the errors implicit in back extrapolation. Certain broad correlations emerge between blood pressure and pulse rate on the one hand and levels of blood volume and amounts of blood loss on the other but the wide range of results showed that no particular combination of signs or symptoms suffice to diagnose the extent of bleeding or even that bleeding has occurred. Even less can single observations of the clinical state give any indication of the extent and rate of further bleeding likely to take place either with spon-

SIGNS OF SYSTEMIC UPSET

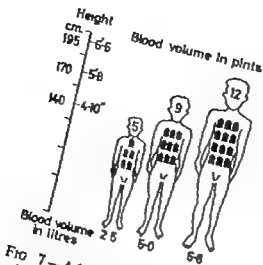


FIG 7—Adult blood volumes by height (By courtesy of the Editor of the *Annals of the Royal College of Surgeons of England*)

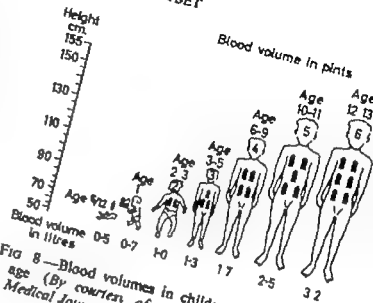


FIG 8—Blood volumes in children by height and age (By courtesy of the Editor of the *British Medical Journal*)

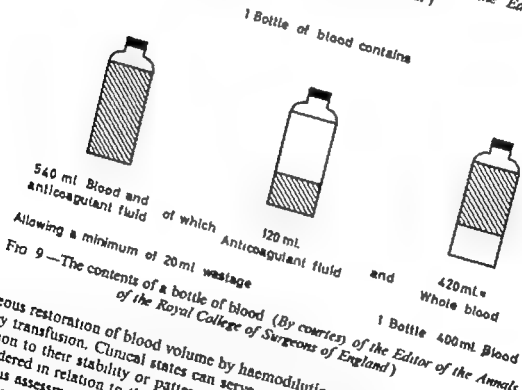


FIG 9—The contents of a bottle of blood (By courtesy of the Editor of the *Annals of the Royal College of Surgeons of England*)

taneous restoration of blood volume by haemodilution or as a result of movement or by transfusion. Clinical states can serve only as guides and then often only in relation to their stability or pattern of change. Their importance must always be considered in relation to the nature and extent of injury and thus involves a simultaneous assessment of the site, nature and extent of bleeding. Variations in normal blood volumes (Figs. 7 and 8), even in previously fit patients, make it necessary to think of clinical states in terms of a proportion of blood volume lost, while the assessment of blood loss by measurement or from the extent of injury requires consideration in terms of absolute volumes. The assessment of transfusion needs must end up in bottles of blood. Here moreover it is

important to recognize that the normal pint (540 ml) of banked blood contains about 120 ml of preservative fluid and only just over 400 ml of whole blood (Fig. 9). The difference can be important. When the objective is to restore red-cell volume it is often wise to ignore the extra volume of fluid. When transfusion is massive and rapid the volume of anticoagulant fluid needs consideration particularly in view of the possibility of citrate overloading (Firt and Hejhal 1957). In the treatment of oligæmia it is therefore necessary to think in terms of percentage and absolute blood loss. It is further necessary to consider bleeding as a continuing process, except when it is known to have stopped, but continuing intermittently rather than evenly.

The process of diagnosis and treatment requires continuous revision of the evidence and an attempt to interpret findings in terms of physiological response. Even for blood loss and blood volume our quantitative measurements are crude but the knowledge so derived can sharpen our clinical wits and make judgment more reliable.

PLANNING TREATMENT

Priorities

Sometimes transfusion though important, may have to be preceded by more urgent measures such as tracheotomy and bronchial suction or immediate closure of a sucking wound of the chest. Local severe bleeding from a wound requires early attention, occasionally with a tourniquet, rarely by direct action. Some workers claim that an occasional patient with obvious major bleeding, reaching hospital *in extremis* may require immediate arterial transfusion.

Routine planning

Usually there is time for a review of the situation before any emergency action is taken. This includes first an evaluation of the systemic and cerebral circulation. In the presence of blood loss or interference with respiratory function it is often difficult to determine whether a disturbance of consciousness is due to a traumatic or other cerebral lesion, or to the cerebral effects of ischaemic anoxia from oligæmia.

A quick review of the patient's general condition is followed by a preliminary estimate of the local injuries. Within a few minutes it should be possible to see the nature of the immediate problem and the degree of urgency with which the next steps must follow.

In severe injuries it is often wise to look at the patient before taking a history but a description of major bleeding at the site of the accident or of the state of the patient immediately after injury can be most important. A clear history of purely local injury may help to distinguish between a vasovagal attack and established oligæmia.

When the patient's general condition is good and there is no evidence of major blood loss, it is possible to proceed with detailed investigation without undue haste. Once a baseline is established the blood pressure and pulse rate should be recorded at 15-minute or 30-minute intervals along with any change in colour, skin temperature, level of consciousness, pain, anxiety or distress, respiratory difficulty, sweating, vomiting, and other symptoms or signs. The emphasis may vary but it has

ASSESSMENT OF BLOOD LOSS

become evident that in multiple injuries early symptoms and signs often fail to indicate lesions that later become decisive.

In the less seriously injured patient whose general condition is good there is no urgency to start transfusion but as soon as it appears that transfusion will be needed, blood should be taken for grouping and cross matching so that subsequent delays can be minimized.

In the presence of obvious bleeding, severe limb or trunk fractures suspected abdominal injuries and in collapsed patients with probable blood loss, blood should be taken immediately for grouping and cross-matching even when transfusion itself can await further assessment.

In the more seriously injured patient an early decision must be taken whether to start transfusion immediately or after blood grouping or a provisional cross-match (see Chapter 6). Sometimes it is advisable to set up an intravenous drip so that there will be no delay in finding veins when it is decided to transfuse. At this stage 5 per cent glucose solution rather than normal saline solution should be used since salt loading may be contra indicated. In the presence of major injuries or established oligæmic collapse it is necessary to plan each movement of the patient so that added trauma is reduced to a minimum, certainly until the blood volume has been increased. Clothes must often be cut off. Emergency splints may be necessary to fix a broken bone even before the patient is turned to examine the back for bruises, wounds, abrasions swelling or tenderness. Movement for examination or for radiology may need to be deferred until transfusion has improved the patient's general condition.

When immediate treatment is not obviously indicated, careful observation during a period of 15-20 minutes without any interference which might change the clinical state for the worse often affords valuable information. A falling blood pressure or a rising pulse rate will usually mean that treatment cannot be delayed.

At some time within the first 30 minutes following arrival at hospital a careful assessment of blood loss must be made.

ASSESSMENT OF BLOOD LOSS

Basic information on the extent of blood loss associated with injuries of various types has been derived from blood-volume studies. The value of blood volume estimates during acute circulatory upsets after injury has not yet been defined but their accuracy in more stable states is sufficient for practical purposes. The biggest source of error is that the patient's initial blood volume and red-cell mass is unknown and may vary by $\pm 10-20$ per cent of the mean normal for height or weight. Nevertheless the evidence derived from blood-volume studies fits in with other haematological investigations (Topley and Clarke, 1956) and with clinical attempts to assess and measure bleeding following trauma. The chief value of blood volume studies has been to indicate the order of blood loss in different kinds of injuries in people of different ages. Transfusion with whole blood based on such evidence has been successful in reversing and preventing clinical circulatory disturbances and in preventing the development of significant anaemia, provided that attention has been paid to the danger of continuing bleeding.

The amount of blood loss can be assessed initially from (1) the presence or

history of visible bleeding (2) the nature and extent of injury (3) the measurement of bleeding at operation including the volume of blood removed from an abdomen or aspirated from a chest (4) in the case of the limbs and to a lesser extent the trunk, from the volume of swelling (5) the general reaction of the patient and (6) blood volume studies. To the knowledge so derived from the individual patient must be added knowledge and experience derived from blood-volume studies in others with similar types of injury.

Subsequent assessments of blood loss can be based on (1) observation of further bleeding from wounds or into dressings (2) increasing swelling (3) increasing signs of abdominal or thoracic damage and (4) haemoglobin determinations. With more detailed investigation the nature and extent of other lesions are unveiled, sometimes only at operation and further clues are given by changes in pulse rate, blood pressure, skin colour and temperature.

Even in the absence of any evidence of further bleeding, continuing bleeding can be expected from many kinds of injury sometimes the amount is clinically predictable and can be covered by transfusion. The contribution of transfusion itself to the extent and timing of further bleeding is of great importance.

External bleeding

External bleeding from injuries can rarely be measured directly. At operation it can be measured in the suction bottle or by swab-weighing. However people can be trained to make approximate estimations from the blood-staining of dressings and clothes and from blood shed on the floor, stretcher and so forth (Clarke and Fisher 1956). Assessment of external blood loss from the appearance of a wound is inherently difficult but a review of patients with open fractures on whom repeated blood volume estimations have been carried out and limb swelling measured or photographed, suggests that external blood loss within the first few hours is usually of the same order as blood loss into the tissues in the majority of civilian injuries with open fractures compound from within. External blood loss has often stopped by the time the patient has reached hospital even from major wounds with moderate sized blood vessels divided. Oozing may continue slowly thereafter even through dressings, or may start again briskly following movement or transfusion, or spontaneously. Bleeding from wounds of the hand, face and scalp is frequently much greater than is often thought. Such patients may collapse while awaiting operation, or under anaesthesia, because the extent of oligæmia has not been appreciated. This can usually be prevented by transfusion and early operation to arrest bleeding.

Bleeding from fractures of the face and base of skull is often concealed by swallowing and considerable amounts of blood can be lost.

Closed bleeding

In attempting to correlate blood loss estimated from blood volume studies with the nature and extent of injury Grant and Reeve (1951) used the hand as a measure of the volume of injured tissue: the closed fist for deep injuries and the open hand for extensive surface injuries. Their information was derived from autopsy as well as clinical material. They graded wounds into small, moderate, large and very large. Small wounds of less than one hand were found to lose less than 20 per cent of total

ASSESSMENT OF BLOOD LOSS

blood volume, moderate wounds of 1-3 hands between 20 and 35 per cent large wounds of 3-5 hands between 30 and 40 per cent, and very large wounds 40-50 per cent. The method is, of course, crude. Estimations of blood volume were extrapolated back from blood volumes at a later stage and did not sufficiently allow for bleeding that occurred between admission and the blood-volume estimation. Neither did the estimations allow for bleeding to be expected subsequently during operation and in the post-operative phase. Nevertheless the attempt was useful and stimulating.

In civilian injuries particularly fractures and large bruises of the limbs it has been instructive to measure the degree of swelling on the assumption that in the early stages most of it is blood (Clarke Topley and Flear 1955). This was done by comparison with the normal limb either by immersion and measurement of water displacement, or by calculation from a series of circumferential measurements at regular intervals over the swollen area and the corresponding segment of the normal limb. The amount of swelling is usually of the same order as the reduction in measurable blood volume. Such attempts at exact measurement are of limited value clinically but a tape measure can be very useful in estimating progressive swelling by repeated circumferential measurements at a few marked points. Training to make assessments of swellings can be facilitated by reconstruction of artificial swellings. The author has found it useful to employ measured volumes of surgical felt which have then been photographed in position on the body for demonstration purposes (Clarke and Fisher 1956) (Figs 10 11 and 12).

Once the expected amount of bleeding from various kinds of fractures and associated soft tissue injuries is known, it becomes possible to assess the probable degree of blood loss from radiological examination of fractures. This is particularly valuable in trunk injuries where the volume of swelling cannot be measured although the extent of blood loss may subsequently become evident from widespread subcutaneous extravasation.

Extent of haemorrhage related to certain injuries

It is possible to put forward a range of amounts of bleeding likely to be associated with particular types of injury

Fractures of the calcaneum fracture dislocations of the ankle and severe sprains — Moderate to severe swelling around the foot and ankle is likely to be associated with primary swelling and haemorrhage of about $\frac{1}{2}$ -1 pint (0 25-0 5 l.).

Closed fractures of the leg — Moderate swelling may be associated with the loss of 1-2 pints (0 5-1 l.)

Fractures of the shaft of the femur — These are commonly associated with swelling and haemorrhage of 1-4 pints (0 5-2 l.) or more

Severe closed injuries of the leg and knee — These can lose as much as 4 pints (2 litres) at the acute stage

Severe fractures of the femur — With gross displacement and swelling these can lose very much larger amounts of blood into the thigh.

Closed injuries of the forearm — With moderate swelling these can account for 1-1½ pints (0 5-0 75 l.)

Closed injuries of the arm and shoulder region — These injuries are occasionally associated with a haemorrhage of 4 pints (2 l.) occasionally more



FIG 10—Measured volume of surgical felt in position on the forearm to simulate swelling due to blood loss of 1 pint ($\frac{1}{2}$ L) (By courtesy of the Editor of the British Journal of Clinical Practice)

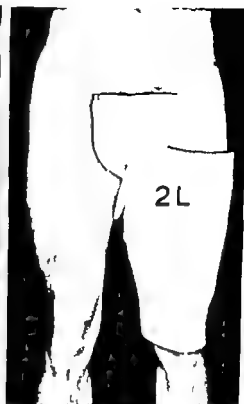


FIG 11—Measured volume of surgical felt in position on the thigh to simulate swelling due to blood loss of 4 pints (2 L) (By courtesy of the Editor of the British Journal of Clinical Practice)

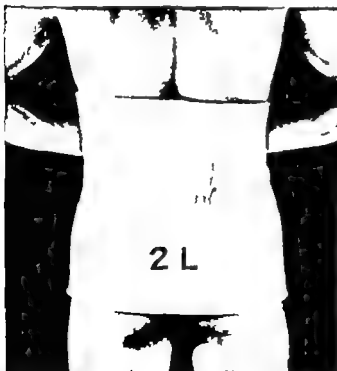


FIG 12—Measured volume of surgical felt in position on the back to simulate swelling due to blood loss of 4 pints (2 L) (By courtesy of the Editor of the British Journal of Clinical Practice)

ASSESSMENT OF BLOOD LOSS

Fractures of the pelvis—These are often associated with very large blood loss but the amount varies with the extent of the bony lesion and associated soft tissue injury

Fractures of the spine multiple fractures of the ribs and severe bruising of the trunk—Even without gross intrathoracic lesions, 2-4 pints (1-2 l) can be lost without clinical evidence of blood loss or special vascular damage.

Open wounds—Blood loss in open wounds varies with the type of injury and the region involved. Wounds of the scalp face and hands can lose a large amount of blood quickly whilst crushing injuries of the extremities even with gross tissue damage, can be associated with little blood loss. When main vessels of the limbs are damaged haemorrhage is extremely variable and cannot be assessed from the nature of the injury without evidence or a history of the amount of initial bleeding.

Continued bleeding

Continuing bleeding into dressings can sometimes be assessed by direct observation. Continuing bleeding into closed limb injuries is common but its extent depends on the nature of the space into which it can occur in addition to the size of the vessels torn and the success or failure of natural or assisted haemostasis. The bigger the segment of the limb affected and the more the soft tissue damage the greater the possibility of severe and continuing bleeding. This is most dangerous in multiple injuries and fractures of the femur and pelvis where with transfusion it is by no means unusual for patients to lose an amount of blood equal to their total blood volume or more.

Blood volume studies

The use of blood volume studies for clinical assessment of haemorrhage at the acute stage is not only limited by the range in the "expected normal" values but also by their possible inaccuracy in states of altered circulatory physiology the time necessary for their completion and the fact that facilities for such investigations are not yet widely available.

Where diagnosis is difficult and particularly when there is no direct evidence of internal or external bleeding yet systemic signs suggest it is possible, a single blood volume determination may be extremely useful provided its potential inaccuracy is recognized. When full replacement therapy has been carried out following multiple injuries and haemorrhage, it is sometimes valuable to have a check on the blood volume once circulatory stability has been achieved. When bleeding is continuing repeated blood volume studies can be helpful in assisting the management of transfusion.

Haemoglobin levels

Serial haemoglobin studies combined with blood volume determinations have shown that the anaemia of trauma is associated with a low red-cell volume and that haemodilution continues over a period of days. The most important factor leading to the reduction in red-cell volume and a low haemoglobin level is haemorrhage. In the majority of severe injuries this haemorrhage continues into dressings and into tissues over a period of hours and sometimes days. Anaemia due to excessive

haemodilution is rare and in the absence of significant infection the haemoglobin level reached 5-7 days after injury provides a useful indication of the extent of blood loss and its replacement (Topley and Clarke 1956)

Haemoconcentration is rare, except in burns severe abdominal injuries with peritonitis and the occasional genuine crushing injury to a limb with the minimum of tissue tearing. Changes in the haematocrit or haemoglobin level during the first few hours are of limited value except when unexpectedly low values suggest a pre-existing anaemia which may increase the need for transfusion but suggest caution in its rate of administration.

Occasionally a rapid fall in haemoglobin over a period of 2-3 hours may indicate extensive internal haemorrhage with little evidence to suggest its origin for example in a patient with a ruptured spleen and paraplegia or a head injury

Effect of transfusion on bleeding

The bigger the injury and the greater the primary blood loss, the more the extent of bleeding will depend on treatment as well as the initial anatomy of the lesion. Comparison of blood volume results in a number of moderately injured untransfused patients with the results obtained in comparable transfused patients suggests that when bleeding is about 20-35 per cent of normal blood volume the total haemorrhage is not significantly increased by transfusion. It is admitted that a patient with moderate injuries may stop bleeding without transfusion and may subsequently bleed less but to adopt this policy would mean an increased risk of death from continuing haemorrhage and of many of the effects of oligæmia discussed in other chapters. With major injuries adequate transfusion will increase the total bleeding but the alternative is often an early death.

With transfusion of about 4-7 l there has been neither a tendency towards a bleeding diathesis nor special difficulty in controlling bleeding. The patient who has lost 40 per cent of his blood volume and is rapidly transfused will bleed more but if transfusion is continued fast enough to raise the blood volume surgical control of haemorrhage is often possible and the chances of survival will increase. Definitive treatment of the individual lesions then becomes possible.

Clinical patterns

Fisher (1958) classified the patterns of circulatory response to trauma into "normal", "tachycardia", "hypotensive" and "hypertensive". There is a general tendency for a rapid pulse rate and low blood pressure to be associated with major bleeding. Sustained systolic pressure below 70 mm. Hg combined with a pulse rate of over 120 per minute in a patient who is pale, cold and sweating usually indicates a haemorrhage of 40-50 per cent of the total blood volume. Such patients nearly always require a transfusion equal to their total blood volume to restore the circulation.

On the other hand, slower bleeding amounting to 30-35 per cent of total blood volume may not be associated with a clinical circulatory change: the patient may be pink and warm with a normal pulse rate and a normal blood pressure.

A raised blood pressure following injury is often associated with minor bleeding (10-20 per cent) but it may be seen in the absence of haemorrhage. The diagnosis of oligæmia is made more difficult by the common superposition of a vasovagal

INDICATIONS FOR BLOOD TRANSFUSION

attack. The patient may present with a low blood pressure, pallor, coldness and sweating, a picture frequently indistinguishable from severe oligæmic shock. This may originate through psychogenic factors. It may develop early or late and be associated with minor or major bleeding. It can only be diagnosed with certainty and disregarded from the point of view of oligæmia or hæmorrhage when the injury is known to be trivial and the patient recovers spontaneously and rapidly.

To assess bleeding or continuing bleeding, changes in the circulatory signs are of more value than the description of a static clinical pattern (Fisher 1958). In the absence of any other explanation, a rising pulse rate, a falling blood pressure or a falling pulse pressure indicate progressive hæmorrhage or that some other factor is altering the response of the organism to hæmorrhage. At times even a blood pressure rising above normal may have this implication. On the other hand, such changes can result from peritoneal irritation, rising intrathoracic pressure from hæmorrhage or a tension pneumothorax. When there is evidence to suggest the presence of damage to the contents of the cranial, thoracic or abdominal cavities the diagnosis of hæmorrhage from physical signs becomes increasingly difficult. Injuries to the spinal cord are frequently associated with widespread sympathetic paralysis and a low blood pressure may result. The fully developed picture of extreme pallor, air hunger, rapid pulse and low blood pressure should usually be assumed to be due to hæmorrhage. In this connection it is important to appreciate that lesions of the cranial or thoracic contents are not contra-indications for transfusion.

INDICATIONS FOR BLOOD TRANSFUSION

Transfusion is indicated following hæmorrhage in trauma for the treatment of established oligæmic shock, for the prevention of oligæmic shock to make anaesthesia and surgery safer, to prevent a number of possible sequelæ and complications of oligæmia, and to modify the illness of trauma.

A fit young adult can certainly afford to lose 1-1½ pints (0.5-0.75 l.) of blood without any need for this to be replaced by transfusion.

Beyond this point transfusion is advisable and the amount required is greater the more serious the injury, the greater the need for major surgery and the greater the likelihood of further bleeding.

With hæmorrhage of as little as 2 pints (1 l.) even in fit young adults, transfusion may be indicated either when further blood loss is expected, anaesthesia and surgery are required, or the injury is likely to predispose to infection.

Precise indications for blood transfusion may depend on the individual opinions of surgeons but more important is the availability of enough blood of the right group and of an efficient organization within the hospital for blood grouping and cross-matching. Most fit adults will survive the loss of 3-4 pints of blood but may subsequently become quite ill. With larger loss of blood, transfusion is always indicated. In severe oligæmic shock rapid transfusion is essential.

There is still a difference of opinion about the objectives and handling of transfusion in severely injured patients. The author believes that in all moderately and severely injured patients the aim should be to replace the whole amount of blood lost by rapid transfusion and then to continue transfusion to cover further hæmorrhage. When this is done the risks of collapse are reduced, anaesthesia and surgery

are safer convalescence is more rapid and uniform and there is some evidence that wounds heal better

When the primary transfusion has been inadequate, the need for late transfusion is indicated by a falling haemoglobin value

Where full facilities for quick and safe transfusion are not available, the transfusion policy must be modified. When haemorrhage is moderate and it is necessary to wait several hours for blood plasma or dextran can be useful in counteracting oligaemia. Their value is limited by the fact that the blood is diluted and because by starting further haemorrhage they may further reduce the haemoglobin level and oxygen-carrying power of the blood even while maintaining the blood volume

Rate of transfusion

The worse the clinical state, the greater the evidence of blood loss and the shorter the time since injury the more rapidly can blood be transfused up to restoration of a normal blood volume. Thus, rather than the mere return to a given level of pulse or blood pressure, should be the objective. Rapid transfusion means the introduction of a bottle (0.5 l) of blood in about 4-5 minutes. It is often possible to transfuse 10 bottles of blood into one vein in less than an hour.

Two or more drips may be necessary if transfusion is not producing an improvement or when further bleeding may be taking place at a rate equal to or greater than that of transfusion. Transfusion should be given through the largest Guest cannula that it is possible to insert, preferably 13-gauge but in some severely injured patients it may be preferable to insert a polythene catheter into a main vein or to cannulate a large vein at the root of a limb. In severe haemorrhage and established oligaemic shock there should be no hesitation in starting a second transfusion whenever there appears to be difficulty with the first.

Venous spasm may be a difficult problem. Sometimes it may be overcome by local heat or the injection of procaine into the drip but the latter has its own dangers of convulsions and hypotension. The rate of transfusion may be accelerated by raising the height of the drip-stand. Alternatively the pressure may be increased by a bellows, and some advocate a rotary pump. When the latter are used it is necessary to insert a filter to prevent contamination from the bellows or the outside air. It is also necessary to remember the danger of air embolism. When a pressure transfusion is indicated the severity of the patient's condition warrants the continuous presence of a doctor but it is the responsibility of every one present to watch the transfusion bottle so that there is no risk of air under pressure entering a vein.

Occasionally with patients suffering from major haemorrhage and arriving at hospital *in extremis* there may be a case for retrograde intra arterial transfusion. In general this method has little advantage over the intravenous route. Firt and Hejhal (1957) have suggested, against a background of experimental evidence, that massive ultra rapid transfusion with citrated blood can lead to spasm of the coronary and pulmonary arteries so that the effect of citrate loading might be confused with apparently irreversible shock. They claimed that the danger of citrate intoxication is reduced by simultaneous injection of calcium gluconate and procaine into another vein. It is probable that the rate of transfusion required to produce this effect is rarely indicated even for severe injuries.

SURGERY AND TRANSFUSION

Signs of full replacement

In peripheral or localized injuries the total transfusion volume may be based on the known extent of injury and bleeding. In more diffuse and deeper injuries it is easier to underestimate than overestimate the total requirements. When in doubt it is wise to err on the side of giving too much rather than too little.

The author has observed that the state of the peripheral circulation can give valuable indications when watched over the whole period of resuscitation. With transfusion the nose and ears become warm the latter pink, at an early stage. The toes become warm and pink last and this usually indicates approaching full replacement. In the absence of respiratory difficulty of any kind with a normal blood pressure and warm extremities visible veins in the neck above the level of the manubrium sternal may indicate a blood volume near to normal.

Transfusion may still need to be continued to cover further bleeding.

Late indications for transfusion

In World War II transfusion of the anaemic patient with established infection was an invaluable aid to the treatment of the infection. States of chronic sepsis could largely be controlled and wound healing stimulated by restoration of the haemoglobin to a near normal level. It was generally considered that a haemoglobin level of 60 per cent of normal in the presence of infection or a major wound was an indication for transfusion. It would appear logical to prevent the development of such anaemia, and when this is done the systemic effects of wound infection can be prevented or brought rapidly under control by antibiotics and surgery.

In the presence of major wounds or significant visceral injuries a falling haemoglobin level after the immediate resuscitation phase is an indication for transfusion. The longer this is delayed the more it may be necessary to consider transfusion with packed cells rather than whole blood. This applies particularly to the aged.

Children and young adults stand blood loss better than those in middle and old age whilst the elasticity of their circulatory systems reduces the danger of overtransfusion. In the elderly and in patients with severe disturbances of lung function transfusion should be given cautiously. Thus also applies to patients with major haemorrhage in whom treatment has been delayed and the plasma volume may have increased. The older the patient and the more severe the systemic illness the more he is in need of early and adequate transfusion but the amount and the speed with which it is given must not overload the circulation.

The first urine specimen obtained after transfusion should be sent to the laboratory for examination for evidence of haemolysis. The empty blood bottles should be kept unwashed for 48 hours so that the remaining blood is available for investigation should a transfusion reaction be suspected.

With a properly organized blood transfusion laboratory service (see Chapter 6) and a service available night and day the dangers of transfusion under emergency conditions can be reduced to a point where they are no greater or less than the dangers of general anaesthesia for a minor operation in skilled hands.

SURGERY AND TRANSFUSION

Resuscitation by transfusion is often the prelude to early surgery and it is usually possible to restore the blood volume before surgery is started. On occasions surgery

RESUSCITATION AND TRANSFUSION IN SEVERE INJURIES

is an essential part of resuscitation either for the control of haemorrhage or for the prevention of complications that can lead to rapid deterioration or early death. The most important indication for early surgery is continuing haemorrhage when there is a possibility of bringing this under control.

Early surgery is necessary to repair tissues in open wounds and in many closed injuries, but when injuries are extensive or multiple it is often wise to delay surgery for an hour or two to allow a further period of observation or to await full resuscitation. It is likewise necessary at times to limit the amount of primary surgery or to reduce the anaesthetic time by simultaneous operations by several teams. The surgeon in charge must take a decision on the state of the patient as a whole. Decisions on priorities may be difficult. Many procedures normally carried out within a few hours of injury can be delayed for hours or days, even occasionally the surgical treatment of wounds unassociated with severe muscle damage. Such delays are made safer by the early use of antibiotics.

Anaesthesia

The risks of general anaesthesia in patients with very severe injuries have been greatly reduced by improvements in blood replacement and advances in anaesthesia (Wolfson, 1959). Nevertheless, in patients with very severe injuries the risks of a general anaesthetic can be excessive; it may be advisable to consider the use of local or regional anaesthesia.

Post-operative observation

Grant and Reeve (1951) stressed the vital need to continue resuscitation and observation during operation and well into the post-operative stage. This is of extreme importance in major and multiple injuries. Continuing or recurring bleeding may lead to sudden deterioration. Important lesions are very easy to miss in the presence of multiple injuries, head injuries or collapse. Their significance may only become manifest later. Charted records should be kept, and repeated visits by members of the medical staff may be necessary to watch for new developments.

It is particularly important to record urine output and to keep specimens of urine for analysis if required. In most injuries where whole blood loss has been adequately replaced there is no need to continue an intravenous drip once bleeding has ceased and the circulatory state is stabilized, since patients will soon be able to drink and eat. They should be progressed as rapidly as possible on to a light diet. In the unconscious patient with head injury tube feeding may be necessary. In a proportion of abdominal injuries continuous gastric suction may need to be associated with intravenous administration of fluid and electrolytes. For the first 24 hours fluid and glucose are the chief needs. When the patient's circulatory state is rapidly stabilized and the alimentary tract is functioning, the electrolytes will usually look after themselves and the patient can soon be given a high protein, high vitamin diet. When normal feeding is not possible sodium and potassium loss must be replaced.

ANCILLARY METHODS OF RESUSCITATION

Oxygen is not indicated for haemorrhage unassociated with interference with respiratory function. The head-down position may be advisable when haemorrhage

ANCILLARY METHODS OF RESUSCITATION

is severe. Once the blood pressure is restored and the patient is conscious he should be nursed flat or on his side unless a chest injury necessitates his being sat up. Sitting up is normally not advisable until transfusion has restored the blood volume to normal or near normal.

Major fractures of the long bones should be splinted during the resuscitation phase and this is often best done by means of plaster slabs, except for the femur where the Thomas splint is invaluable. Wounds should be covered and minor or moderate bleeding controlled by bandaging over plenty of gauze and cottonwool.

There is no logical case for heat in the treatment of "shock". Hot cradles and hot water bottles are not only unnecessary but may be dangerous. Heat produces vasodilatation which drains blood away from essential organs and may produce a state of collapse. It may also increase fluid loss by sweating. When the skin is pale and cold it acts as a more efficient insulator so that the body (rectal) temperature may rise. The shocked or unconscious patient is easily burned by hot water bottles or cradles and the physical signs of systemic or local disturbances of the circulation are masked by artificial warming. The temperature of the shock room and operating theatre should not exceed 68 F and may with advantage be cooler provided that it is comfortable for the staff. Blankets are unnecessary and the patient is best covered with only a sheet.

Fluids by mouth are contra indicated so long as there is any likelihood that anaesthesia will be needed. In any case the severely injured patient is likely to vomit and thirst is best relieved by restoration of blood volume to normal by transfusion.

Cooling and hibernation

The role of definitive cooling in severe injuries has not yet been clearly defined. In head injuries control of hyperthermia is certainly indicated and the temperature is best kept between 96 and 98 F. Chlorpromazine in small doses may be beneficial in patients with head injuries associated with excessive neurogenic reactions leading to hypertension, tachycardia, or an increased respiratory rate, provided that transfusion is continued. All drugs, including chlorpromazine, should be used cautiously as we are not yet fully aware of the extent to which they can obscure the symptoms and signs of undiagnosed injuries. Any more elaborate programme of hibernation can only be legitimately considered if it is clear that there is no alternative line of treatment. In the presence of pain, morphine or pethidine sometimes potentiated by chlorpromazine are indicated but should be given in relatively small doses intravenously so that their effect can be observed and dosage controlled accordingly.

Noradrenaline

The use of noradrenaline or other vasopressive drugs is rarely if ever indicated in the presence of oligaemia. These drugs do not increase cardiac output, but they increase the work of the heart and, most important, they produce prolonged renal ischaemia and greatly increase the danger of renal failure.

There is much room for further scientific evaluation of the role of many methods of treatment in addition to transfusion but their use at the present time remains largely empirical.

REFERENCES

- Blalock, A. (1940) *Principles of Surgical Care Shock and Other Problems* London Kimpton.
- Cannon W B (1919) *Spec Rep Ser med. Res Coun. Lond.*, No 25 109
- (1923). *Traumatic Shock* New York Appleton.
- Clarke R., and Fisher M R. (1956) *Brit J clin. Prac.*, 10, 746.
- Topley E. and Flear C. T G (1955) *Lancet* 1 629
- Flit, F., and Hejhal L. (1957) *Lancet* 2, 1132.
- Fisher M R. (1958) *Clin. Sci.* 17 181
- Grant, R. T and Reeve, E. B (1951) *Spec Rep Ser med Res Coun., Lond.*, No 277
- Pickering, G (1958) *Brit med. J.*, 2, 1117
- Topley E. and Clarke R. (1956). *Blood* 11 357
- Wolfson, L. J (1959). *Anaesthesia for the Injured* Oxford Blackwell

ORGANIZATION AND PROBLEMS OF EMERGENCY BLOOD TRANSFUSION

CHAPTER 6

S SEVITT

TRANSFUSION of blood is important in the treatment of many injured patients some of whom require 5 10 20 or even 30 pints of blood over a period of hours. It should generally begin as soon as possible and may need to be started within minutes of admission to hospital. Every transfusion carries a risk the risks are multiplied when the transfusion is large and may be further increased under emergency conditions particularly when a number of casualties are simultaneously treated. Only careful organization of a hospital transfusion service and constant vigilance by those who operate it can reduce the hazards to the minimum.

Emergency transfusion is now possible because the combination of efficient donor services and the ability to preserve blood for up to 3 weeks by acid-citrate dextrose solution has made the blood available. In Great Britain and other countries the donor-supply organization is separate from the hospital transfusion laboratories and is run through a regional system of blood transfusion services. This separation has many advantages and a similar division should operate when a donor service has to be organized in the present chapter.

The great majority of haemolytic reactions and almost all fatal ones follow ABO or Rhesus (Rh) incompatibility sometimes technical mistakes are responsible but more often the errors are clerical or distributive. Safe transfusion can almost always be achieved with blood of the same ABO group and Rh (D) type as the patient (homologous blood) after compatibility tests between the patient's serum and the donor cells. The clinical urgency may determine whether homologous transfusion and cross matching are possible or not, and if not, the cloth has to be cut to meet the urgent requirements. One or two bottles of O blood may have to be given before the results of blood grouping allow homologous transfusion. Some homologous blood may have to be given without cross-matching or after a provisional compatibility test.

THE BLOOD BANK

The refrigerator should be specially designed for a hospital blood bank (Sevitt 1953). An even temperature of $3-5^{\circ}\text{C}$ adjustable by external control is required throughout the cabinet. Supercooling must be eliminated and it should be impossible to freeze the stored blood. Defrosting should not be necessary and the cooling system (preferably an ice water mixture surrounding the walls of the cabinet) should be so designed that there is a hold-over period of 12-24 hours should the refrigerating mechanism fail. This can be very important at night. A continuous temperature recording apparatus and an alarm bell should be fitted.

the latter to ensure that the cabinet doors are properly closed after removal of blood at night.

Blood stocks

No rules can be laid down for the number of bottles of O Rh positive and Rh-negative and A Rh positive and Rh negative blood which should be stored for emergency use as this depends on the turn-over from planned transfusion, the frequency of emergency transfusion and on the speed and regularity with which stocks can be replaced by the donor service. A quarter of the bottles used in the Birmingham Accident Hospital are given to emergency cases normally our stock consists of 6-10 bottles of O Rh positive blood a similar number of A Rh positive bottles 2-4 A Rh-negative bottles and a minimum of 4 bottles of O Rh negative blood but further supplies can be obtained quickly from the regional service

ORGANIZATION AND TEAM WORK

Personnel

The transfusion laboratory should be under the direction of an experienced pathologist or haematologist who should always be available for consultation on technical and medical matters. He should plan and organize the technical procedures the keeping of records, the training of technicians, investigate transfusion reactions and keep the department up to date by periodic trials of new techniques. Only exceptionally should a clinician be charged with the running of a hospital transfusion service and then he should receive special training. The routine technical work should be carried out either by trained technicians or junior pathologists or by a combination depending on local circumstances. All results must be checked by someone else and the work of junior technicians must always be supervised by an experienced person. Technical and clerical mistakes are not to be permitted and this means *inter alia* that sufficient staff are necessary to prevent hurrying and harassing through pressure of work in a busy department.

The time has passed when blood grouping and allied procedures were part time occupations for resident clinical staff. They should not be expected to carry out blood grouping or cross matching tests at any time least of all in emergencies at night or week-ends when all their attention should be devoted to the patient. Under the former system the inevitable errors were responsible for most of the blood transfusion reactions which then occurred and which are now known to be avoidable. Blood grouping and cross-matching techniques are full of potential difficulties not apparent to the inexperienced clinician.

Team-work

The hospital transfusion laboratory is the key to and link between the service supplying the blood and hospital staff who order it for a patient only a close liaison can achieve integration of the whole service and the smooth transfer of safe blood along the line to the patient. Agreement must be reached on the manner of transfer of information blood samples, donor bottles and other details and must be understood and operated by the technicians, resident medical and nursing staff. Degrees of urgency must be defined. When difficulty arises a word of explanation may prevent a delay in transfusion and often saves time and temper. The non

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medical technician must not be rushed or harassed by house surgeons who must restrain the authority conferred by a medical qualification

The patient's blood sample

A venous sample (10 ml if possible) should be transferred to a sterile centrifuge tube as soon as it is suspected that transfusion of the injured patient may be necessary. Early collection may save time later. The tube must be labelled clearly with the patient's name, hospital number and the date. Labelling errors are one of the common causes of incompatibility and cannot be detected by the laboratory. Unlabelled or improperly labelled tubes are a menace; the technician should be instructed to refuse them. The doctor personally must label the tube before he leaves the bedside and preferably just before he withdraws the blood. Occasionally blood cannot be obtained from a badly shocked patient because of intense venoconstriction; collection may then have to be delayed until one or two bottles of O blood (or plasma) have been transfused.

The blood-transfusion request form

A request form designed for blood transfusion work and containing a section relating to degrees of urgency should always accompany the blood sample to the laboratory. The written information must include the patient's name (if known otherwise a temporary pseudonym), hospital number, sex and age, the number of bottles of blood believed to be needed and an outline of the patient's injuries. It should indicate the degree of urgency that is the time when the first bottle or bottles will be required, and if and when an operation will be performed. If possible the questions of pregnancy, previous miscarriages and history of previous transfusion should be noted. Reference to previous admission to hospital may be important since a quick search of the card-index file in the laboratory may lead to special information. According to the information available the patient is classified into a "good risk" or "bad-risk" recipient.

"Good-risk" and "bad risk" recipients

Bad-risk recipients are those who may have developed from transfusion pregnancy or disease an immune antibody which might react with transfused cells. Good risk recipients are those without a history of haemolytic disease; previous transfusion, pregnancy or miscarriage for them the cross-matching procedure need not be as elaborate as in the "bad risk" cases.

Degrees of urgency

Full compatibility tests take up to 2 hours to perform, which is usually too long before beginning transfusion of a seriously injured patient. Degrees of urgency are useful to define since they indicate to the clinician what laboratory tests are possible and to the technician what is expected of him. The following division has been found useful.

(1) *Immediate transfusion*—No delay is possible and the first 1-3 bottles will have to be O preferably Rh negative. It is usually possible to ensure that subsequent bottles are homologous ABO and Rh type.

(2) *Maximum delay 15 minutes*—ABO grouping is always possible and Rh-typing generally possible, but not cross-matching, so that transfusion can commence with homologous blood which has not been subjected to a compatibility test. Subsequent bottles should be provisionally or fully cross-matched blood if time permits

(3) *Maximum delay 30-45 minutes*—Blood grouping, Rh typing and provisional cross-matching tests can be carried out so that transfusion can commence with homologous blood subjected to provisional compatibility tests

Collection and delivery of donor bottles

Only 1 or at most 2, bottles of blood should be released at a time for one patient, torrential haemorrhage or very severe blood loss excepted. Unofficial sub-banks in other hospital refrigerators should not be substituted for regular collections of bottles from the official hospital bank. An important detail is the collection of the correct bottle from the blood bank and its delivery to the correct patient. The donor bottles must, of course be labelled with the patient's name, hospital number and ward, but labelling may not prevent the wrong bottle being issued or a bottle being delivered to the wrong patient, perhaps with the same name. These human errors are more possible in emergency work and can be only avoided by constant vigilance and double checking at each transfer. Our practice is to use a "blood-bottle request form" for each bottle issued. The patient's name, hospital number and ward (or department) are entered, the form is signed by a doctor or nurse and is given to the messenger collecting the bottle. At the blood bank the patient's details on the labelled bottle and the request form are checked by the technician and the messenger. At night the technician may be absent then the bottle should be collected only by a doctor or a nurse who should enter the serial number of the bottle removed, sign the form and leave it in the laboratory for information. At the bedside the labelled identity of the bottle should be checked against the identity of the recipient by two persons, generally a nurse and a doctor. Nurses are accustomed to a double check when drugs like morphine are to be given. How much more important it is to double check the identity of a bottle of blood.

The 24-hour emergency laboratory service for blood transfusion

Modern society and the tradition of medicine demand that a patient injured at night should be as effectively treated as one injured during day time so that the transfusion service should operate efficiently day and night, seven days a week. The organization necessary will vary with local circumstances. Some laboratories will prefer to man the "out-of-normal hours" service through a roster of laboratory technicians on duty or on call, some will prefer to use resident pathologists whilst others will combine the services of both. Ideally the technician should be on duty within the hospital and reside there overnight. Alternatively he should be on call at home with telephonic communication and rapidly available motor transport provided. This generally delays our service for 20-40 minutes and though not ideal it is found to be adequate for nearly all emergencies. Transfusion of the casualties who cannot afford the extra wait is begun with un-cross-matched O Rh-negative blood.

The long term success of an emergency service manned by technicians depends

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not only on selection and training but also on the frequency of duty and on payment. Nobody should be overloaded with duty periods as this will surely lead to a breakdown in the service or the person concerned. Adequate extra payment should be made to technicians in the author's opinion preferably on a seasonal basis. Die hards may try to analyse the cost of an emergency service in terms of pounds and shillings per patient or per bottle of blood they should be reminded that cost per incident is not the basis of the fire brigade or life boat services.

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Meticulous ABO grouping and Rh (D) typing of the patient's blood is the first essential and the necessity of avoiding errors must be emphasized. The technician must not carry out simultaneous tests on samples from two patients as this is a potential source of clerical error. All grouping and typing performed outside normal laboratory hours should be repeated the following morning. For information on blood group serology and techniques related to transfusion additional to that which follows, the reader is referred to Medical Research Council Memorandum No. 36 (1958) Dunsford and Bowley (1955) Mollison (1956) and James (1958). The first step is to remove with a pipette about 1 ml of bloody serum from the blood sample into a small test tube on which the patient's name is written. The serum is quickly separated by centrifuging and the deposit is washed once by resuspending in saline and centrifuging again.

ABO grouping

Slide or tile methods are not completely reliable sooner or later a false diagnosis of AB through excessive rouleaux formation is made or A is confused with B. The multiple tube technique should always be used in laboratory practice, except when mass casualties make it impracticable (see page 83). With experience the test can be completed in 2-3 minutes. It is in two parts: the recipient's cells and serum are tested separately against known antisera and red-cell suspensions respectively at room temperature: the results of one act as a check on the other.

The test

Six 3-inch-by-1/2-inch test tubes in a rack are consecutively labelled "anti A", "anti-B", "anti AB", "A cells", "B cells" and "auto" respectively. The labelling refers to the known reagent added. (The "auto" tube is used to detect non-specific autoagglutination.) One or two drops of about a 5-per-cent suspension in saline of the patient's cells are added to the first three tubes and the last one. One drop of anti A serum is added to the first tube, 1 drop of anti B to the second, and 1 drop of anti-AB (from an O person) to the third. The antisera must be avid and potent and tested daily against known cell suspensions. The third (anti AB) tube checks the results of the first and second tubes and facilitates the diagnosis of weak A subgroups, since agglutination of these cells is often stronger with anti AB sera than with anti A sera. One or two drops of the patient's serum are placed in each of the last three tubes. Two drops of a known A group red-cell suspension (approximately 5 per cent and preferably A subgroup) are added to the fourth tube and similarly two drops of B suspension to the fifth. The known A and B cell suspensions are freshly prepared each day and kept in the refrigerator ready for use. All six tubes are gently shaken to mix and are centrifuged for 1-1 minute at about 1 000-2 000 revolutions per minute. They are replaced in the rack and shaken gently to resuspend the deposit. The group is determined by the reaction of the patient's

cells to the three antisera. Agglutination is gross and unequivocal and is read macroscopically. The fourth and fifth tubes determine the presence or absence of anti A and anti B agglutinins in the serum and the reciprocal relationship of these to the II and A cell groups is used to check the patient's group. Any abnormality in the expected relationship must be checked as a possible error and if necessary the test is repeated but this is rarely necessary. The cell suspension should then be washed once more in saline and other batches of anti-A and anti-II sera opened.

This method has been used for more than 20 000 routine and emergency groupings at the Birmingham Accident Hospital and has given speedy and completely trustworthy results.

Rhesus (D) typing

The conventional tube method of Rh typing generally takes 1-2 hours at 37°C. to complete and is unsuitable for emergency work but it should always be performed as a check on the quick method. Most of the anti D sera available are relatively weak and many agglutinate Rh positive cells only when suspended in a colloid medium like albumin.

Two of the various techniques devised to overcome these drawbacks are described below and can be recommended. Agglutination is speeded by modifying the environment of the cells since the antigen antibody reaction itself is quick. Positive reactions are nearly always obtainable within 10 minutes and results are quicker with stronger sera. Positive and negative control tests with O Rh positive cells and AB Rh negative cells respectively should be set up at the same time.

Occasionally Rh typing is doubtful the patient must be regarded provisionally as Rh negative and transfused with Rh negative blood. If the doubtful result turns out to be Rh positive but due to the modified D-antigen known as D^u it is wiser to regard the recipient as Rh negative because anti D antibody formation has been reported after transfusion with Rh positive blood.

The slanted capillary tube method

This method was described by Chown and Lewis (1946-1951). The patient's cells are allowed to fall through a narrow column of antiserum in a capillary tube of bore 0.4-1.0 mm. inclined at about 45 degrees. In a positive test the red cell column breaks up into multiple small segments. The test is wholly reliable when saline-agglutinating Rh anti-D sera are used and when the test cells are washed and suspended in saline.

The capillary tube (about 8 cm. long) is dipped into the serum, allowing about 2 cm. to fill by capillary traction. The same end of the capillary tube is then dipped into a 20-30 per cent suspension of the patient's cells in saline and, avoiding an air lock, a column of red cells about 2 cm. long is allowed to enter. The serum end of the capillary tube is stuck into plasticine or sealed with Vaseline and the tube is inclined at an angle of 45 degrees. It may be placed in the incubator at 37°C. or more conveniently a rack made of Perspex is used. This is lighted and warmed by an electric bulb placed behind an opal Perspex panel. Granular segmentation of the column of red cells is usually detectable in about 4-6 minutes and definite by 8-10 minutes. A thin unsegmented column is a negative reaction. False positives (serration of edge without segmentation) may occur if incomplete anti D sera (albumin-type) are used and this is not recommended.

The slide "sandwich" method

This method was described by Stratton (1955). The special advantage is that excellent results are obtained with albumin type antisera. A suspension of red cells is mixed

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with bovine albumin and the antiserum and is sandwiched between two glass slides. This speeds agglutination and facilitates microscopy

Single volumes (approximately 0.03 ml.) of antiserum, 30 per cent bovine albumin and packed, washed test cells are placed near the middle of a glass slide. A marked Pasteur pipette is used for standard volumes. The three reagents are mixed over about two-thirds of the slide area. A second slide is laid on top of the first and with the red cell serum, albumin mixture in between, forms the sandwich. The sandwich is incubated at 37°C. for 10 minutes and examined microscopically. It is advantageous to press the slides gently during microscopy to produce a moving film.

Occasional false negative tests are said to occur but not false positives.

Cross-matching tests

The dual purpose of cross-matching is to eliminate incompatibility from errors in the ABO grouping of the donor or patient and to detect the presence of unusual antibodies in the patient's serum active against the donor cells at body temperature

Antibodies against human red cells vary in their properties. Some like anti A and anti B antibodies agglutinate cells suspended in saline or a colloid medium like albumin whilst others, so-called incomplete antibodies produce agglutination only in a colloid medium (the incomplete anti Rh anti D is the most important of these). In saline suspension the globulin antibody is adsorbed on to the cell surface but produces no apparent effect but when an anti human globulin anti serum is added to cells washed free of protein the cells are clumped. This anti globulin test (indirect Coombs test) is very sensitive and some antibodies like the Duffy antibody (anti Fy^a) cannot be demonstrated in any other way. Other sensitive tests employ proteolytic enzymes like trypsin or papain which so alter the cell surface that agglutination takes place in saline when the antiserum is added. No single test is wholly reliable and the full battery which can be performed on a single donor sample is considerable but some are unnecessary in good risk cases. In a busy department a line has to be drawn between those which are essential as routine procedures and those which need to be done in bad risk cases and in special investigations

Our routine cross-match consists of three tests (1) saline agglutination test (2) albumin agglutination test at 37°C. and (3) the indirect Coombs test. The enzyme tests supplement the others only when they are specially indicated. The suggestion of Sachs (1942) that the sensitivity of the saline cross-match with fresh serum may be increased if the cells are suspended in 2-3 per cent saline solution should be pursued whilst the simplified one-stage trypsin-saline test described by Young (1958) may be a useful addition.

Our emergency cross-match is so related to routine unthurned practice that the former may proceed to the latter and the latter may be interrupted at two stages. The emergency cross-match is thus divided into two grades: minimal and further tests. The minimal cross-match consists of saline and albumin tests examined after at least 15-minutes incubation whilst the further test is the indirect Coombs test. Samples of donor blood removed from the pilot tubes attached to the bottles are washed at least once in saline as a prelude to cross-matching.

Saline test

Equal volumes of the patient's serum, saline solution and 10 per cent donor cells in saline are placed in a tube.

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Albumin test

The saline content must be low and a "split drop" (half volume) of an approximately 50 per cent donor-cell suspension in saline is added to 2 volumes of patient's serum and 2 or 3 volumes of 30 per cent commercial bovine albumin.

These tubes set up for each donor sample, are placed in the 37°C. waterbath. Unhurried incubation lasts 1-1½ hours and in emergency it takes at least 15 minutes. The tubes are centrifuged for about 1 minute, replaced in the waterbath for 2 minutes and examined microscopically. If no agglutination is seen the bottle of blood is released but the tubes are replaced in the waterbath for the full incubation and then re-examined.

Indirect Coombs test

If time permits this test should be done before the bottle is released. It may be carried out on the cells in the saline cross match test but it is preferably set up as a separate test. If possible the test should not be performed before 30-minute incubation of the serum, red cell, saline mixture. After incubation the cells are washed thoroughly 3 times by centrifuging, packing and resuspending in saline to remove every trace of human serum. Two drops of anti-human globulin serum are added to one drop of washed cells (approximately 20 per cent suspension) on a white tile or in a small test tube which is centrifuged after 5 minutes. This part is done in duplicate using two different dilutions of the anti human globulin serum to avoid a prozone inhibition phenomenon. The absence of agglutination indicates a compatible cross-match. Positive and negative controls should be set up with each batch of tests by adding the anti-human globulin serum to washed-sensitized cells (usually O Rh-positive cells sensitized in saline suspension by an albumin-type of anti D serum) and to washed AB, Rh-negative cells respectively. The control suspensions should be prepared daily and kept ready for use in the refrigerator.

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Various problems occur but only the two most important can be considered here (1) The Rh-negative recipient and (2) transfusion with large volumes of un-cross-matched O blood. In addition a word is needed on the transfusion aspects of the mass-casualty problem.

Rh-negative recipient

For clinical purposes the Rhesus problem can be considered in terms of Rh positive and Rh negative patients and donors because the Rh D antigen characteristic of Rh positive cells is the most likely of all the Rh antigens to stimulate the production of antibody. Ideally all Rh-negative recipients should be transfused only with Rh negative blood but in practice this is not always possible, particularly with O recipients. Group O Rh negative blood is in short supply because priority is given to the emergencies of women in labour who have the special risk of possible immunization by pregnancy.

The risks to Rh negative recipients from Rh positive blood vary and three degrees of importance have to be recognized.

Incompatibility due to Rh antibody in the recipient

This is the most important risk because of the immediate danger of a severe and possibly fatal haemolytic transfusion reaction from the destruction of the transfused Rh-positive cells. The antibody has nearly always been produced

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through previous transfusion with Rh positive blood or through immunization during pregnancy by a Rh positive foetus. Next to ABO errors Rh incompatibility is the commonest cause of severe haemolytic transfusion reactions.

Men and women found to have a Rh antibody must never be transfused with Rh positive blood, with one proviso. No patient should be allowed to die of haemorrhage because Rh negative blood is not available.

Danger to future pregnancies

The development of a Rh antibody in a female after a transfusion with Rh-positive cells may produce a miscarriage or erythroblastosis foetalis in a subsequent Rh positive foetus. Thus every effort should be made to give Rh negative blood to all Rh negative girls and women of child bearing age particularly if the woman is childless. In the absence of Rh negative blood the risk of immunization and damage to future pregnancies by transfusion of Rh-positive blood has to be weighed against the clinical danger.

Risk from future transfusion

In other patients the hazard following Rh immunization is possible incompatibility in future transfusions with Rh positive blood. This may be important in those likely to need subsequent transfusions and in all young people but it is less important in elderly people and in those with a short expectation of life from carcinoma or other causes. Rh negative blood is desirable but not essential. The serum of all Rh negative recipients given Rh-positive blood should be examined for an irregular antibody during the following weeks. The first examination should be at 7-14 days but immunization is often slow and may not be detectable for 2-4 months. When it occurs Rh positive cells rapidly disappear from the circulation but no clinical effect develops unless Rh positive blood is transfused again.

Transfusion with un-cross-matched O blood

In civilian hospital practice emergency transfusion of O blood should either be unnecessary or be restricted to the first bottle or two by which time ABO grouping will have been carried out. homologous transfusion can then follow. Under "field conditions" in civilian practice (and for battle casualties) ABO grouping is not safe or practicable and the giving of un-cross-matched O blood obviates the risk of ABO incompatibility. Persons with group O blood have long been recognized as universal donors because there is no ABO incompatibility between the recipient's serum and the donor cells. Group O transfusions are generally safe as witness the hundreds of thousands of successful un-cross-matched transfusions given in the Spanish War during World War II and in the Korean War. In the majority of A and AB recipients the transfused anti A and anti B agglutinins are quickly diluted and neutralized and little or no damage occurs to the recipient cells. There are two exceptions to this general rule: the transfusion of large quantities of O blood and the giving of blood from a "dangerous universal donor".

Wasteful transfusion

When large volumes of O blood say 10-20 bottles are given to an A patient his capacity to dilute and neutralize the transfused anti A agglutinins is limited and

anti-A can be detected in the recipient's plasma. Generally the anti A is weak and persists for only a few hours but sometimes it is moderately strong or may be detected for a few days or both. The anti A acts on the recipient's cells, a proportion of which are destroyed and removed from the circulation. The total haemoglobin level falls and serial red cell counts after differential agglutination reveal a relative increase in the proportion of O-donated cells in the blood. Evidence of intravascular haemolysis is absent or slight haemoglobinuria is rare but may occur whilst an acute haemolytic jaundice, usually subclinical develops. The serum bilirubin level may rise rapidly to about 3-5 mg. per 100 ml but biliuria is absent. The latter distinguishes the episode from that of hepatic jaundice which may occur in injured patients after blood transfusion (Sevitt, 1958). The direct Coombs test (anti globulin test) on the post transfusion cells may be slightly positive owing to fixation of the transfused anti A agglutinin on circulating recipient cells. The reaction is generally subclinical or mild. If it is relatively serious for example if haemoglobinuria is found it is likely that one or more of the O donor bottles was from a "dangerous universal donor" (see below).

Two interrelated problems may arise. First, there is usually no pretransfusion sample of the patient's blood and it may be difficult to be sure of the patient's ABO group from the post transfusion sample and impossible to determine the Rh type of the recipient when much Rh-positive blood has been given. Secondly if further transfusion is necessary should it be O blood or blood of the homologous group if that is A, B or AB? Transfusions of 10-30 bottles of O blood to an injured adult will replace most or virtually all the recipient cells by donor cells, and post transfusion grouping may be very difficult. For example, if the patient is group A only a few small cell clumps may develop after adding potent anti A serum to a blood sample. Other evidence of the correct group should then be sought. Blood from a haemothorax or haematoma should be examined as this may contain a higher proportion of recipient cells. Supplementary evidence may be obtained by the weak and transient nature of the anti-A agglutinin compared with the anti B agglutinin in the post transfusion serum and by testing the patient's saliva for A and B secretor activity. The choice of blood for further transfusion should depend on laboratory findings. If there is reasonable doubt of the patient's true group O blood must be continued. If the patient is found to be group A the blood chosen depends on the presence or absence of circulating anti-A antibody and on evidence of sensitization of recipient cells. Group O blood should continue to be given as long as there is evidence of anti A in the plasma (the tests should be carried out in saline and albumin suspensions both at room temperature and at 37°C. and the indirect Coombs test performed) or as long as the direct Coombs test shows evidence of fixation of antibody on some of the cells. When these tests (and the cross-matching tests) are negative homologous A blood should be given. Each case should be studied carefully and treated on its merits. Our experience is based on a clinico-pathological study of over 20 casualties given O blood in large or relatively large volumes by a surgical emergency team sent to small peripheral hospitals or to the site of a serious accident: the patients were then transferred to the Birmingham Accident Hospital. The general advice of Crosby (1955) that O blood should continue to be given for 2 weeks after massive O transfusion is too sweeping. Homologous blood is preferable when it can be safely given because the continued transfusion of O blood maintains a difficult situation and continues the extra

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vascular destruction of recipient cells including, as Crosby and Akeroyd (1954) showed newly formed reticulocytes

Transfusion should be continued with Rh-positive blood unless only O Rh negative blood had been given originally and the patient is Rh negative

Dangerous universal donor

A proportion of O donors differ from the majority in the character of the anti-A in the plasma, which may provoke an acute haemolytic episode in A or AB recipients. The major differences from the usual iso-agglutinin is that fresh unheated serum can haemolyse A or AB cells at 37 C. (*alpha*-haemolysin) and that the agglutinin titre is generally higher than 1 in 200. In general the characteristics are those of an immune antibody. The blood of these dangerous universal donors must be reserved for O recipients and not given to A or AB subjects. The problem to the hospital transfusion service has diminished because the diagnosis of 'dangerous universal donors' has rightly become the responsibility of the supply service. Such bottles should be labelled 'for O recipients only'.

Transfusion and the mass-casualty problem

Consideration will not be given to conditions of war which are a special problem in this nuclear age but will be confined to peaceful civilian life in which railway and other disasters occasionally provide a large number of casualties. A minority require resuscitation before they can be transferred to hospital whilst the majority can be moved safely provided that transfusion is set up soon after their arrival. The transfusion supply services of a region must be planned to cope with these occasional incidents and co-ordinated with emergency medical teams' supplies of O blood must be sent quickly to the scene of the accident for use by the emergency teams. In these circumstances blood grouping should not be attempted.

When the stream of injured patients arrives in hospital blood for transfusion may be required for 20-40 or even more of them. This imposes two demands on the transfusion laboratory: one of supply and the other of quick and safe ABO grouping. The former can be met only by effective liaison with the regional blood supply service. Since the successive ABO groupings have to be quick and the possibility of clerical error within the laboratory eliminated, the technique may have to be amended to suit the exceptional demand. The method advocated by the author depends on the possession of an avid high titre hyperimmune anti-A serum which can coarsely agglutinate a suspension of A cells within 10-20 seconds. The test is a 'one-serum' method and anti B serum need not be used. One drop of a 10-20 per cent saline suspension of the patient's blood is placed in a tube labelled with the patient's name and a drop of the potent antiserum is added. The result divides the patients into two groups, A positives (A and AB) and A negatives (O and B). The former are given A blood and the latter O blood. This makes use of the supply of A blood in the bank and is preferable to O transfusion of all the patients. If a hyperimmune anti A serum is not available the tube should be centrifuged for half a minute before reading the test.

As soon as the rush is over all groupings must be checked by the multiple tube method and Rh typing performed. Cross-matching should begin when all the more serious cases have been provisionally grouped and their transfusion set in motion.

REFERENCES

- Chown, B., and Lewis, M (1946) *Canad. med Ass J.*, 55 66
- — (1951) *J clin. Path* 4 464
- Crosby W H (1955) *Milit Med.*, 177 354
- and Akeroyd, J H (1954) *Blood*, 9 103
- Dunsford, I and Bowley C. C. (1955) *Techniques in Blood Grouping* Edinburgh Oliver and Boyd.
- James, J D (1958) *Practical Blood Transfusion* Oxford Blackwell
- Medical Research Council Memorandum No 36 (1958) *Determination of the ABO and Rh (D) Blood Groups for Transfusion*. London H M Stationery Office.
- Mollison, P L. (1956). *Blood Transfusion in Clinical Medicine* 2nd ed. Oxford Blackwell
- Sachs, H. (1942) *Lancet* 1 473
- Sevitt S (1953) *J clin Path* 6, 324
- (1958) *Brit J Surg.*, 46, 68.
- Stratton, F (1955) *Brit med. J.*, 1 201
- Young, N A. F (1958). *J clin Path.*, 11 311

CHAPTER 7

TRAUMATIC URAEMIA

S SEVITT

INTRODUCTION

INTEREST in post traumatic uraemia was aroused by the description of fatal cases of severe oliguria among air raid casualties in London and elsewhere during World War II (Bywaters and Beall 1941 Bywaters and Dible 1942 Dunn, Gillespie and Niven, 1941) Previous accounts were then rediscovered including those relating to casualties of the Messina earthquake and of trench warfare in World War I the review of Minami (1923) and the description of Husfeldt and Bjerring (1937).

The clinical picture during the air raids was that of a patient who had been trapped under fallen masonry. A limb or other part of the body was severely crushed—hence the term crush anuria after rescue the affected part became greatly swollen clinical shock developed and the patient was transfused with plasma or whole blood. In spite of general improvement the flow of urine decreased to a trickle and the patient became uraemic and died. The urine was coloured brown by myohaemoglobin released from areas of crushed ischaemic muscle. Myohaemoglobin was put forward as the cause of the oliguria but the hypothesis lost ground when, on the one hand, myohaemoglobinuria in injured patients without renal damage and, on the other, severe oliguria after injury without excretion of pigment were reported.

Renal failure soon became recognized as a complication of a variety of non-operative conditions including various kinds of injury severe haemorrhage post water fever, extensive burns abortion, sulphonamide intoxication black lyte imbalance incompatible blood transfusion, dehydration, diabetic coma, electro-lyte imbalance surgical operations pulmonary embolism and paralytic ileus and from various bacterial infections and poisons including uranium salts carbon tetrachloride carbon monoxide, mercuric chloride, salicylates and various cresols. The list extends with investigation and experience. From being a rare condition acute renal failure is now regarded as one of the most important disorders affecting the kidneys.

Nevertheless a common clinical syndrome following such varied conditions does not necessarily mean identical pathogenesis, functional disturbance or morphological changes. For this reason the non-committal terms traumatic uraemia or acute renal failure are used here synonymously rather than terms like lower nephron nephrosis or acute tubular necrosis which are no more than histological descriptions and which may confuse the issue. This is particularly necessary at the present time because recent attention has been drawn to features which make the present tubular view of pathogenesis unacceptable. The description of non-oliguric uraemia (Sevitt, 1956a) has shown that the oliguric syndrome is only a part,

albeit a prominent part, of the picture of acute renal failure. Moreover there is now evidence that tubular function continues to be active or hyperactive rather than impaired (Graber and Sevvitt 1959, Sevvitt, 1956a) so that tubular damage cannot determine the composition of the urine and the concept of tubular necrosis as the basis of the uraemia cannot be accepted. A modified concept of pathogenesis is presented here the central theme of which is a persistent fall in glomerular filtration rate (Sevvitt 1959).

EARLY AND DELAYED URAEMIA

Acute renal failure may develop at any time after injury but its onset is most frequent during the early 'shock phase'. It is convenient to divide the cases according to the time of onset into early and delayed forms. The former are intimately related to acute changes in circulatory haemodynamics precipitated by trauma and blood loss whilst the latter are associated with the circulatory effects of certain complications especially paralytic ileus, electrolyte disturbances and pulmonary embolism. Of course, early and delayed effects may merge and supplement each other in some patients particularly those with abdominal injuries. In this way a subclinical renal state may be changed into a progressive and severe condition.

Prophylaxis

An important by-product of the policy of early transfusion and adequate replacement of blood loss has been the welcome experience of a reduced incidence of early traumatic uraemia. This has become an unusual, almost rare complication in the Birmingham Accident Hospital after limb and trunk injuries and this is attributed to the early arrival of injured patients in hospital and a policy of energetic blood replacement. A large proportion of the small number of cases now seen follow severe abdominal injury where complications like vomiting, paralytic ileus, dehydration and electrolyte imbalance play predisposing parts. Other cases follow head injury. Some are precipitated by a pulmonary embolus in these cases the circulatory changes which produce a prolonged period of hypotension are no doubt of importance. Clinically it is important to recognize this type because anticoagulants are an essential part of therapy many instances can be prevented by prophylactic anticoagulant therapy (see Chapter 17). The delayed cases in recent years have become more apparent now that the shadow of uraemia in civilian traumatic practice is fading. In burned patients on the other hand, there is still a relatively high incidence of early uraemia even though intravenous and oral fluids are energetically administered.

OLIGURIC AND NON-OLIGURIC URAEMIA

Acute renal failure should now be classified broadly into oliguric and non-oliguric forms. The division is clinically important because severe oliguria produces the danger of water retention, hyperkalaemia, hypocalcaemia and acidosis but it is a little arbitrary because there is considerable range of urine output in different

OLIGURIC AND NON-OLIGURIC URAEMIA

patients and perhaps at different times in one patient. A short period of early oliguria may be followed by a normal polyuric output by continued oliguria or by irregular phases of normal reduced or even polyuric flow. Severe oliguria has received most attention because it is easily diagnosed and is dangerous to life but it is only a part of the spectrum of acute renal insufficiency. Indeed the upper limit of oliguria is difficult to define. Some workers regard 300 ml. per day as the maximum (Bull, Joekes and Lowe 1949) others 400 ml (Swan and Merrill 1953) 500 ml (Lucké 1946) Teschan and his colleagues, 1955) or even as much as 1 l. per day (Muirhead and Stirman 1952).

Uraemia with severe oliguria

The syndrome of uraemia with severe oliguria is now well known and need not be elaborated. Oliguria (typically 50-200 ml. per day) is established within 6-36 hours after injury and persists or commences in spite of transfusion and a restoration of blood pressure. Many patients die from other complications of injury during the first two or three days and before uraemia develops in others the general condition deteriorates after four or five days and symptoms of uraemia, including those of hyperkalaemia, hypocalcaemia and acidosis appear. The general illness may be worsened by water logging from excess fluid therapy by anaemia and by bacterial infection. The sodium and chloride levels of the plasma fall perhaps to 120 mEq and 80 mEq per l respectively because of a redistribution in the body. Azotaemia is considerable the blood urea level reaches 200-800 mg. per 100 ml and the plasma creatinine 5-20 mg. per 100 ml depending on the duration of the disorder and on the excess of post traumatic (protein) catabolism. The urea and creatinine clearances fall often to only 5-10 per cent of normal reflecting the low rate of glomerular filtration. The urine is acid in reaction the specific gravity is generally 1010-1016 proteinuria is common and the deposit contains numerous casts most of which are granular and often pigmented but some contain epithelial or red cells. Initially the urine may be pigmented by free haemoglobin in burned patients or from myohaemoglobin after muscle damage in injured persons but in traumatic cases pigment is often absent. True haematuria is slight but common. The electrolyte composition of the urine is considered below. In brief the urine often shows the usual post traumatic pattern of low sodium and chloride and increased potassium concentration (Fig. 17) thus differing from that reported by Bull, Joekes and Lowe (1950).

Diuresis

The oliguria generally lasts 7-10 days but may be shorter or longer and occasionally lasts 3-4 weeks. It is followed either by a sharp increase in output up to perhaps 5 l. per day or more commonly by an increase over a few days to a normal or slightly polyuric output, particularly when fluid intake has been restricted. Azotaemia may temporarily worsen before it improves. The urine at this stage may contain large quantities of sodium, potassium and chloride alleged to result from faulty tubular reabsorption. This explanation is unlikely because *inter alia* the excess of potassium must result from a high rate of tubular excretion. The negative balance of sodium and chloride is more often the response to previous overloading than tubular impairment. Prolonged salt wasting is a myth and the

high output is maintained by continued over administration in the belief that the kidney is unable to conserve salt. Excretion is increased if the large amount of salt passed the previous day is given. If this is repeated daily high salt excretion is maintained and the kidney is frustrated in its attempt to attain a normal salt and water balance (Merrill, 1955a). If hypokalaemia occurs through excessive loss of potassium a potassium salt should be given.

During the later phase of diuresis azotaemia disappears and the urine flow if previously high is restored to normal levels. The renal blood flow and glomerular filtration continue to improve but some renal impairment may persist for months.

Non-oliguric uraemia

Attention has recently been drawn to uraemia with little or no oliguria after burning and injury (Sevitt, 1956a) and reference has been made to the condition in battle casualties (Teschner and his colleagues 1955) and following head injury and major surgery (Taylor and Reid, 1956). More recent experience has shown that it may also follow other civilian injuries particularly abdominal trauma and also incompatible blood transfusion. It is at least as common as and probably more common than the oliguric form, but it is likely to be overlooked.

Azotaemia develops in spite of resuscitation but the urine flow is relatively normal. There is a gross fall in glomerular filtration but little or no oliguria because the tubular reabsorption of water diminishes sufficiently to balance the reduced amount filtered by the glomeruli (Graber and Sevitt, 1959). The urine flow may be disturbed by an early transient oliguria and by phases of polyuria. In many cases the azotaemia is apparently irreversible by transfusion or other therapy and the blood urea rises to 200–800 mg per 100 ml. before death or recovery occurs. In others the azotaemia is temporary and relatively moderate: the blood urea rises to 100–200 mg per 100 ml. and then falls to normal within a week or so. In burned patients temporary azotaemia mainly occurs in children and the progressively irreversible state in adults thus may reflect a youthful haemodynamic renal resiliency which is deficient in adults. The patterns of change in the concentrations of sodium, potassium and chloride in the urine are similar in both azotaemic and non-azotaemic injured patients. Unlike the oliguric form the excretion of potassium, phosphate, hydrogen ions and water is not limited by a low urine flow: the plasma potassium and phosphate concentration show little or no increase, neither hypocalcaemia nor acidosis are likely to develop and the risk of water logging is slight. Non-oliguric uraemia is not as dangerous as the oliguric form: nevertheless nitrogenous and other end products of metabolism, such as sulphates and phenols excreted by glomerular filtration are retained in the blood and may have clinical effects. The composition of the urine including the presence of granular and other casts is in most cases essentially similar to that found in the oliguric form.

Differentiation from so-called extrarenal azotaemia may be important although the border line is often indefinite and at times may be artificial. A urea-clearance test is useful but if the volume of urine is difficult to measure because of incontinence or for other reasons the principle of the urea-concentration test may be employed since the raised plasma urea level acts in lieu of the oral test load of urea. In non-oliguric uraemia the urine/plasma (U/P) ratio of urea is always below

PRINCIPLES OF TREATMENT

20 and is often only 10-5 or less. In contrast, the U/P ratio of urea in extrarenal azotaemia is more than 20.

PRINCIPLES OF TREATMENT

It is now recognized that oliguric renal failure is not necessarily fatal and that recovery may occur after days or even weeks of oliguria, but the mortality is high, particularly after burning. The object of treatment is to keep the patient alive by reducing water logging, hyperkalaemia, acidosis and azotaemia until the kidneys recover. The conservative regime advocated by Bull, Joekes and Lowe (1949) has been replaced in recent years by more active therapy especially to combat the accumulation of excess water and potassium in the body. The indications for haemodialysis in particular are becoming more clearly defined. Other therapy is also often required, such as blood transfusion to combat anaemia and antibiotics against wound and other infections.

Restriction of fluid intake

In oliguric patients water retention must be avoided. The total volume of all fluids given daily should be restricted to the amount lost the previous day by sensible and insensible means minus an estimate of the cellular water released by catabolism. This is equivalent to the previous day's volumes of urine and vomit plus in a temperate climate 500-600 ml. Daily weighing of the patient is a valuable guide to the amount of water needed and, if therapy is well carried out, the body weight should actually fall a little (about $\frac{1}{2}$ -1 pound) daily because of catabolism. Electrolytes should be restricted to replace those lost.

Azotaemia, diet and steroid therapy

Low-protein feeding is necessary because the end products of nitrogen metabolism cannot be excreted. The high-calorie/nitrogen poor concentrate of a glucose-fat emulsion given by intragastric drip (Bull-Borst regime) (Bull, Joekes and Lowe, 1949) has played a useful role in drawing attention to the possibilities of therapy, but more recently a high-calorie intake has been thought to be less important and 200-400 g. of glucose daily is considered adequate. Most of this is usually given intravenously as a hypertonic (25 per cent) solution, if necessary by a major vein.

Steroid therapy, particularly with testosterone, has been advocated to reduce protein catabolism and promote anabolism; its value has not been established. Nevertheless, this is a growing therapeutic possibility because some success has recently been claimed for a synthetic 19 norsteroid (norethandrolone, Nilevar) in obstetric cases of acute renal failure, although in non-obstetric cases the results were unsatisfactory (McCrudden and Parsons 1958). Encouraging results in chronic renal failure were reported by Gjorup and Thaysen (1958).

Hyperkalaemia

Plasma potassium levels above 8-9 mEq per l are generally dangerous because of a toxic myocardial effect although the correlation between plasma concentration and degree of toxicity is not very close. Electrocardiography may give useful

information about the state of the myocardium Treatment of hyperkalaemia is as follows

(1) Potassium is removed from the extracellular fluid by the alimentary use of cation-exchange resins or more effectively by blood dialysis with an artificial kidney apparatus (see below)

(2) Potassium is deposited in body cells by the administration of glucose and insulin glycogen is formed and potassium is fixed This is less effective and the effect is short lived

(3) The myocardial effect of hyperkalaemia is reduced by calcium therapy because hypocalcaemia enhances the toxic action. The effect is temporary and therapy has to be repeated (see below)

Hypocalcaemia

Uraemic twichings and tetany result from hypocalcaemia which in turn results from hyperphosphataemia. Therapy with calcium salts has to be repeated because the raised plasma inorganic phosphate antagonizes and shortens its effect.

Acidosis

Retention of fixed organic acids produces acidosis which, if severe (plasma bicarbonate below 12 mEq per L) must be treated by alkaline salts (sodium bicarbonate or lactate) given intravenously if necessary Lesser degrees of acidosis should also be treated but not at the risk of producing alkalosis

Blood dialysis

Blood dialysis through Cellophane tubing (artificial kidney) was first described by Kolff and Berk (1944) who used a rotating drum instrument Other instruments in which the blood is under positive pressure have since been developed such as the Alwall Skeggs-Leonard and Kolff disposable-coil instruments these have the added advantage that water can be removed from the blood. Dialysis is the most useful measure in restoring blood chemistry the main indications are high serum potassium levels not responsive to other methods of treatment and considerable overhydration Periodic dialyses temporarily relieve uraemic symptoms by clearing the extracellular fluid of excess potassium phosphates fixed acids urea, creatinine, water and other products of catabolism and by elevating the lowered plasma sodium, chloride, calcium and bicarbonate levels Dialysis should not be delayed unduly and should be begun before the blood urea reaches 400 mg. per cent. Recent developments in mobile units may bring therapy to the patient. For technical and other details see Alwall, 1947 Anthonisen and his colleagues 1956 Hamburger and Richet, 1956 Joekes, Scott and Jackson, 1958 and Merrill 1955b

MORPHOLOGICAL CHANGES

The morphological findings in traumatic uraemia may be broadly classified into tubular necrosis and glomerular changes

Tubular necrosis

The term lower nephron nephrosis (Lucké, 1946) has been too often accepted uncritically and applied to the clinical syndrome as if it were a disease. Various other names including haemoglobinuric nephrosis (Mallory, 1947) "glomerulo-tubulo-nephrosis" (Zollinger 1952) and tubulo-interstitial nephritis" (Brun, 1954) have been used but necrosis of tubules is said to be characteristic (Bull and Dible, 1953; Oliver 1953; Oliver, MacDowell and Tracy 1951) and the picture has been named acute tubular necrosis. This was subdivided into distal tubular and proximal tubular necrosis according to the main site of lesions (Sevitt, 1956b). Distal tubular necrosis was further divided into diffuse and focal lesions according to the histological density.



FIG. 13.—Diffuse distal tubular necrosis after burning. Many Henle tubules contain haemoglobin casts; foci of epithelium are thinned and show pykno-necrosis. Haematoxylin and eosin $\times 300$.

Distal tubular necrosis

Diffuse distal tubular necrosis—The evolution of changes partly depends on the release of haemoglobin or myohaemoglobin—multiple pigmented casts, distending, locally blocking, and producing foci of pykno-necrosis in distal tubules (Fig. 13) are characteristic and are seen in patients dying with incipient uraemia within

TRAUMATIC URAEMIA

2-3 days of extensive burning or haemolytic transfusion reaction (Sevitt, 1956b 1957) or after crush injuries with myohaemoglobinuria. Otherwise the early changes are often slight or absent and this is important in view of the emphasis laid on tubular change. According to Dible (Bull and Dible 1953) the earliest change is a selective necrosis of the descending part of the proximal tubule 30-40 hours after the onset of oliguria. Necrotic lesions then increase (Fig 14) and casts appear in the lower nephron. Pigmented casts are difficult to explain when there is no evidence of intravascular haemolysis or release of myohaemoglobin but close examination may show red cells apparently escaping into tubules through small communications between ruptured venules and tubular epithelium. Focal tubular ruptures involving the basement membrane result in tubulo-venous anastomoses and mural thrombi, particularly in the boundary zone casts are dislocated, tubular urine probably leaks out and interstitial oedema and inflammatory collections of lymphocytes and other cells develop around tubules and veins. Some



FIG 14—Tubular necrosis affecting the wide loops of Henle in a patient with severe oliguria after multiple injuries. Haematoxylin and eosin $\times 210$

FIG. 15—Extensive necrosis of proximal tubules in a burned patient. Haematoxylin and eosin $\times 200$



times the interstitial changes are diffuse and well marked earning the name tubulo-interstitial nephritis. The disruptive lesion, originally described by Dunn, Gillespie and Niven (1941) was termed tubulorrhexis by Oliver MacDowell and Tracy (1951) to distinguish it from so-called simple nephrotoxic necrosis of the proximal tubules within intact basement membranes. Foci of tubulorrhexis are found in the proximal and distal tubules whilst nephrotoxic necrosis is found only in the proximal tubules.

Later changes include bizarre epithelial spreading, abortive repair of the ruptured tubules and perhaps fibroblastic replacement of the interstitial inflammation.

Focal distal tubular necrosis—Tubular lesions resemble those of the diffuse form but are confined to scattered foci.

Proximal tubular necrosis

In many cases the brunt of damage falls on the proximal convoluted tubules which become a sea of dead and dying epithelium within intact basement membrane (Fig. 15). The glomeruli are generally spared and the distal convoluted tubules appear relatively intact. An irregular subcapsular strip of the cortex frequently remains viable as if it were kept alive by the capsular vessels. Sometimes it is very congested and perhaps focally haemorrhagic.

Proximal tubular necrosis is usual in the middle-aged and elderly patients dying with azotaemia after extensive burning (Sevitt, 1956b) or severe injury. Nephrosclerosis may predispose to this because the direct channel between the

afferent and efferent arterioles which develops in many juxtamedullary glomeruli during glomerulosclerosis (Trueta and his colleagues 1947) could increase cortical ischaemia if there was vasospasm of the interlobular arteries

There may be histological evidence of renal ischaemia after severe injury. In some fatally injured patients with fat emboli in the cerebral and other systemic vessels there are few or no emboli in the glomerular capillaries; this strange absence from the heaviest sites of systemic embolism (see Chapter 14) indicates intense ischaemia of the renal cortex. Proximal tubular necrosis is often seen.

Frequency and significance of tubular necrosis

The common association of tubular necrosis with renal failure does not establish it as the morphological basis since association does not prove causality. Not for the first time in renal pathology has the accent on morphological changes led the physiologists astray. The association is often close but it is statistical and not absolute and one can occur without the other. Diffuse distal tubular necrosis and proximal tubular necrosis after burning are usually associated with oliguric or non-oliguric uraemia but this is not invariable: occasionally severe tubular necrosis is associated with moderate functional change or has no functional significance (Sevitt, 1956b). No histological differences are to be seen between the oliguric and non-oliguric kinds. Other studies have shown that oliguric and non-oliguric uraemia after burning and injury can occur without tubular necrosis (Graber and Sevitt, 1959; Sevitt unpublished observations). Brun and Munck (1957) were impressed with the contrast between the moderate structural changes and the severe functional breakdown: they found tubular necrosis in only 5 out of 33 cases of acute renal failure.

On the other hand cases of focal distal tubular necrosis generally have no clinical importance although occasionally the histological pattern is associated with acute renal failure (Sevitt, 1956b). Many battle casualties with histological lesions have died without clinical evidence or suspicion of renal damage. Mallory (1947) found haemoglobinuric nephrosis in 19 per cent of fatal battle casualties whilst Anderson and Steer (1955) found renal lesions in as many as 36 per cent of men dying from war wounds after reaching hospital. This contrasts with a 0.5 per cent incidence of traumatic uraemia among wounded men reaching hospital and 1.6 per cent among major surgical cases in the same battle area (Teschner and his colleagues 1955; Ladd, 1955).

Thus tubular necrosis can occur without renal failure and vice versa: they are commonly associated because they have common pathogenetic factors (see below) (Fig. 19). The important conclusion cannot be escaped that tubular necrosis is not the morphological basis of renal failure. Microdissection studies have shown that even when many nephrons are damaged others are normal (Oliver, MacDowell and Tracy 1951; Darmady 1957) so that when tubular necrosis is present there are two populations of nephrons: damaged and undamaged. The kidney's reserve of nephrons is large and more than three-quarters of the renal tissue has to be removed surgically before there is clinical effect.

Other tubular changes

A hydropic tubular change (so-called osmotic nephrosis) affecting proximal convoluted tubules may be a prominent feature and a hyaline droplet change may

FIG 16—Multiple fine droplets of lipid (fine black dots) in glomerular epithelium in a case of renal failure after burning. Frozen section, oil red O $\times 350$



affect the neck like origins and other parts of the proximal convoluted tubules. Neither of these changes is related to tubular necrosis or degeneration and they are probably osmotic and metabolic effects respectively the former related to therapy with crystalloids like glucose

Changes in glomeruli

The glomerular tufts are said to be essentially normal although Bowman's capsule often contains eosinophilic exudate and globules the parietal epithelial layer is desquamated and later shows cubical hyperplasia.

Glomerular lipid droplets

Recently it has been shown that the kidneys from patients with uraemia after burning often show multiple fine droplets of lipid in the epithelium of the glomerular tufts (Fig. 16), (Sevitt, 1957 Graber and Sevitt, 1959) Multiple droplets are usual when tubular necrosis is present or when azotaemia has been found but their presence and density are not consistently related to tubular necrosis. The significance of the fatty change has not been fully assessed but it is abnormal and may be a histological sign of a more serious lesion. Electronmicroscopy of the

normal glomerulus (Pease 1955) shows that the capillary endothelium is full of fine pores about 0.1μ in diameter and that the epithelial cells have multiple gaps or fenestrations between fine foot like processes cemented to the basement membrane. The pores and gaps must mean that the only structure normally capable of filtration is the intervening basement membrane. It is conceivable that subtle glomerular changes impossible to define by light microscopy may develop in patients with acute renal failure and contribute to the persistence in the fall of glomerular filtration. Changes like acute cellular swelling with obliteration of pores or fenestrations for example might oppose filtration whilst the appearance of fatty droplets might be the histological sign of the more serious lesion not visible by light microscopy. Electronmicroscopy of the glomeruli from cases of traumatic uraemia is awaited with interest.

RENAL FUNCTION

The present evidence indicates that the basic defect in acute renal failure is a low glomerular filtration rate (GFR) precipitated by renal ischaemia from vaso-spasm and maintained by ischaemia and possibly changes within the glomeruli. The emphasis laid on tubular damage and dysfunction as the key disorder is exaggerated.

Renal ischaemia after injury

The work of Lauson, Bradley and Cournand (1944) showed that a considerable renal ischaemia develops in recently injured patients and is due to a selective renal vasoconstriction because the reduction in renal flow (often to 10-20 per cent of normal or less) is greater than the fall in the cardiac output or arterial pressure when these are reduced. Transfusion hardly affects the renal blood flow although the arterial pressure and the cardiac output are restored. These results suggest that renal vasoconstriction after injury begins before the cardiac output and arterial pressure fall and persists for some time after they are restored. None of the patients in this series apparently developed traumatic uraemia but the persistence of renal ischaemia may well be related to that found when renal failure occurs. Renal ischaemia was also found in wounded soldiers (Burnett and his colleagues 1947, Ladd 1955) and in animals subjected to haemorrhage or trauma (Van Slyke and his colleagues, 1944, Keele and Slome, 1945). Ladd (1955) found that mild injuries were followed by a slight reduction in flow, severe injuries by a major reduction and that operation and anaesthesia further increased the ischaemia. Criticism on technical grounds has been made because the renal blood flow was generally measured by the clearance of para aminohippuric acid (PAH). A relatively complete extraction of PAH from the blood by the kidneys into the urine is assumed and this may not occur in injured patients. This criticism cannot apply to Keele and Slome's (1945) experiments in which renal blood flow was measured directly and by a plethysmographic method.

Renal ischaemia in acute renal failure

A low renal blood flow was found in 4 patients with oliguric renal failure following carbon-tetrachloride poisoning (Sirota, 1949) and in 7 patients with

RENAL FUNCTION

oliguric failure of varied aetiology (Bull Joekes and Lowe 1950) using the full technique of P.A.H. injection renal vein catheterization and the Fick principle, thereby obviating the criticism of clearance tests. The blood flow was less than 10 per cent of normal during the acute oliguric phase it then improved but remained low for the first month. A gross renal ischaemia (low effective renal plasma flow) was confirmed by Ladd (1955) in battle casualties with renal failure even though they had received large or very large transfusions of blood.

Recent work has confirmed the reduction in renal blood flow but suggests that the degree of ischaemia may not be so profound. Using a method involving radioactive krypton Munck (1958) found that the renal flow was about one third of normal and much greater than that measured by P.A.H. clearance. The filtration rate was grossly and more considerably reduced. The disproportionate fall in filtration supports the present contention that ischaemia cannot account for the full reduction in glomerular filtration and that other changes must have contributed. Maluf's (1949) finding that intravascular lysis plus dehydration in dogs produces oliguria, azotaemia and a profound reduction in the filtration rate is relevant because after *in vivo* injection of Indian ink via the aorta the glomerular blood flow was not found to be abnormal. Renal vasospasm could not have been responsible.

Low glomerular filtration

The inability to explain traumatic uraemia on the basis of tubular damage makes a reassessment of glomerular function necessary. There is no doubt that the clearance values of inulin and endogenous creatinine are greatly reduced during the early diuretic phase and that the low values are associated with renal ischaemia (Eggleston and his colleagues, 1942; Corcoran and Page, 1943; Lauson, Bradley and Courmand 1944; Bull, Joekes and Lowe 1950; Brun 1954; Ladd 1955). Low clearances have also been found during the shock period following extensive burns and during the subsequent oliguric or non-oliguric renal failure when this occurs (Dziemian, 1948; Graber and Sevit, 1959).

Non-selective reabsorption of the filtrate

Normally inulin and endogenous creatinine clearances measure glomerular filtration rate but this interpretation has been criticized if tubular damage is present. The latter is said to permit unselective reabsorption or back-diffusion of filtered water and solutes and thus, not the low G.F.R. is said to account for the low clearance values (Philips and Hamilton, 1948) and for the oliguria and uraemia (Lucké 1946). This concept has bedevilled all conclusions related to glomerular filtration because it implies that the glomeruli are producing a filtrate but that the rate of filtration cannot be measured by clearance tests. Richards (1929) observations on the nephrons of frogs poisoned by mercury are quoted since he found that glomerular filtration continued but that the filtrate was completely absorbed by the tubules and so no urine was passed but in this experiment the glomerular blood flow continued normally in contrast to the renal ischaemia of traumatic uraemia. Unselective reabsorption might occur but its effect would be limited to damaged nephrons and the result would be a reduction in the number of functioning units which would continue to form urine although handicapped by

a low G F R. Unselective reabsorption can hardly determine the composition of the urine because little if any of the filtrate in the affected tubules would reach the ureter. It cannot be responsible for the low clearance values because the creatinine and inulin clearances in renal failure are equally reduced (Corcoran and Page, 1943; Brun, 1954; Graber and Sevvitt, 1959) which would be expected if they both measured the G F R. This is in contrast to the low and varying ratios of urea to creatinine clearance (Graber and Sevvitt, 1959) which indicates tubular discrimination between urea and creatinine. Moreover the very low sodium and chloride and high potassium concentrations in the urine must result from intense tubular activity and not from unselective reabsorption from tubular damage.

It may be concluded that the endogenous creatinine and inulin clearance values are measures of the effective G F R. that is of that part of the glomerular filtrate which produces the flow of urine.

Temporary and persistent fall in filtration

Serial estimates of the endogenous creatinine clearance after extensive burns show a considerable fall in the G F R within a few hours of burning and the beginning of therapy (Fig. 17) (Graber and Sevvitt, 1959). The fall is often interrupted by considerable fluctuations. Azotaemia develops when the mean of the fluctuating G F R is below 50 per cent of normal for 24 hours. In some cases the G F R returns to normal within a few days but in others the low G F R. (15–20 per cent of normal) persists or worsens and is unaffected by the transfusion therapy in these cases azotaemia is severe and generally progressive (Fig. 17). Some patients in this series had severe oliguria, others a normal urine flow (Fig. 17 right). In the wounded soldiers studied by Ladd (1955) temporary reduction in filtration was related to moderate injuries and a persistent lowering to severe injuries often with abdominal trauma. Transfusion with large quantities of blood did not prevent or restore the severe persistent fall in filtration.

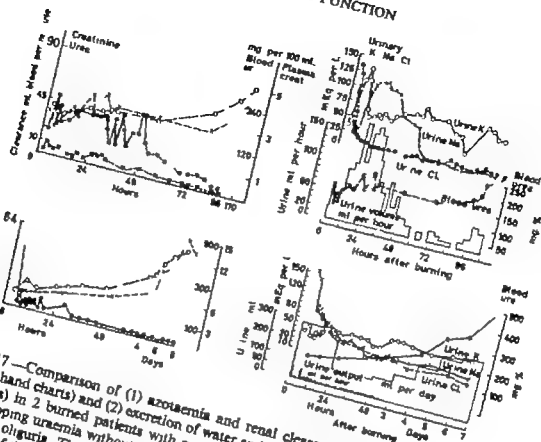
Tubular function

Continued tubular activity is indicated *inter alia* by the renal ability to reabsorb sodium and chloride, and to excrete potassium, and by the absence of glycosuria.

Excretion of electrolytes

The patterns of change after injury in the concentrations of sodium, potassium and chloride in the urine are similar both in those patients who develop severe azotaemia and in those who do not (Sevvitt, 1956a and unpublished observations; Graber and Sevvitt, 1959). There is usually a rapid onset of low sodium and chloride concentrations (1–20 mEq per l.) or a low chloride with intermittent low sodium phases and an acute rise in potassium concentration (up to 200 mEq per l.) followed by a higher than normal potassium level. Essentially similar patterns are usually found in oliguric and non-oliguric patients (Fig. 17 top and bottom right) irrespective of the fall in G F R. or the presence or absence of azotaemia. Low sodium and chloride concentrations in the urine are, of course, due to active tubular reabsorption from the filtrate, and high potassium concentrations are due to active excretion by tubules so that these changes reflect tubular activity. They depend on the adrenocortical secretion of aldosterone and are part of the body

RENAL FUNCTION



17—Comparison of (1) azotaemia and renal clearance of urea and creatinine (left hand charts) and (2) excretion of water and electrolytes in the urine (right hand charts) in 2 burned patients with renal failure. The top charts refer to a patient developing uraemia without oliguria whilst the bottom ones refer to a patient with severe oliguria. The bold numerals on the left charts refer to the expected normal value of the creatinine clearance. Zero is the time of burning.

Note the immediate fall in the GFR (creatinine clearance) followed by a subsequent decline after 48 hours (top left) and in the right hand charts the low chloride, low sodium (in two phases in the top chart) and the high potassium concentrations in the urine of both non-oliguric and oliguric patients (B) courtesy of the Editor of the *Journal of Clinical Pathology*

reaction to "stress" and blood loss (see Chapter 2) The reaction occurs after burning, injury and haemolytic transfusion reactions whether or not severe azotaemia or oliguria develops Tubular ability to reabsorb sodium and chloride and to excrete potassium is indicated by the high plasma urine (P/U) ratios for sodium and chloride (generally 5-30:1) and high U/P ratios for potassium. The tubular reabsorptive ability is more accurately assessed according to Platt's (1950) formula by calculating the fraction of filtered sodium which is excreted in the urine. Values below the normal 2 per cent (often 0.5-0.1 per cent were found) are proof of active tubular reabsorption (Fig. 18) The ability of tubules to differentiate between sodium and chloride is revealed by the short phases of high sodium and low chloride output during which the urine contains large amounts of bicarbonate. Tubular hyperactivity may persist until death supervenes or may recur after stress of subsequent operation or infection and before the patient succumbs Tubular necrosis is often found at necropsy sometimes it is slight or even absent so that

the functional evidence of tubular activity is independent of the morphological state Ladd (1955) also concluded that tubular reabsorption of sodium was normal in uraemic battle casualties whilst Taylor (1957) found high P/U ratios for sodium and chloride in acute renal failure following head injuries and surgical operations

These findings are in contrast to the relatively low P/U ratios of sodium and chloride (generally 2-3:1) reported by Bull Joekes and Lowe (1950) they concluded that the urine approximated to a plasma filtrate and that impaired tubular activity was responsible Their patients and those of Brun (1954) however did show considerable P/U variations from case to case, that for chloride ranging from 10 to 1 Few of their cases were post traumatic and few if any were studied from the time of the clinical incident precipitating the oliguria when the renal response of the adrenocortical reaction to stress might have been very apparent Their finding of a high potassium excretion during the early diuretic phase is indicative of tubular hyperactivity not an inability to conserve potassium since potassium excretion is an active distal tubular process

In brief tubular ability to reabsorb sodium and chloride to excrete potassium and to differentiate between these ions (and other substances) indicates a high degree of persistent tubular power and may be in contrast to the degree of tubular necrosis found

Excretion of water

There is a curious difference between the active tubular reabsorption of sodium and chloride and a reduced reabsorption of water Normally about 1 per cent of the filtered water is passed as urine but in patients with renal failure following burning or injury the fraction excreted progressively rises and may reach 10-15 per cent (Fig. 18) (Ladd, 1955 Graber and Sevvit, 1959) Overall the fraction is inversely related to the GFR and parallels the rising blood urea level A reduced tubular reabsorption of water occurs in non-oliguric uraemia (Fig. 18 top) and when there is severe oliguria (Fig. 18 bottom) whilst the volume of urine passed is dependent on the balance between the low GFR and the reduced reabsorption of water Oliguria is absent when the reduced reabsorption compensates for the reduced filtration and is present when compensation is inadequate Reduced reabsorption of water has been interpreted as an impaired tubular ability (Bull Joekes and Lowe 1950) but then it would be a selective impairment because other absorptive powers seem adequate It might be an osmotic tubular diuresis related to the rising head of urea in the plasma filtrate or it might be mediated by inhibition or antagonism of antidiuretic hormone

Urine concentration and specific gravity

The specific gravity (1010-1016) and concentration (350-450 milli-osmols per l) are low largely because the concentration of solutes, and particularly urea, is lowered by the diminished reabsorption of water from the filtrate and not because of tubular damage as alleged.

Reabsorption of glucose

Glycosuria related to the post traumatic hyperglycaemic response may occur after injury (see Chapter 2) but it generally disappears within 24 hours both in

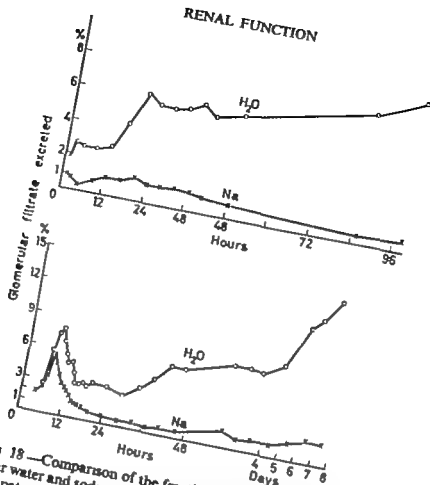


FIG 18—Comparison of the fractions of filtered glomerular water and sodium which were excreted in the urine in 2 patients developing uraemia after burning, one (top) without oliguria and the other (bottom) with severe oliguria. There is a considerable and progressive increase in the fraction of filtered water which reaches the bladder in both patients (diminished tubular reabsorption of water) compared with a very low excretion of the sodium filtered by the glomeruli (increased tubular reabsorption of sodium). (By courtesy of the Editor of the *Journal of Clinical Pathology*)

those patients who develop uraemia and in those who do not. This indicates proximal tubular ability to reabsorb glucose Bull, Joekes and Lowe (1950) agreed that glycosuria was rare and found this difficult to explain on the basis of tubular damage. Glycosuria did appear in patients with renal failure after a loading dose of glucose had been given and they calculated that the tubular reabsorptive power was impaired. In injured patients this would be difficult to distinguish from the common decrease in glucose tolerance but even if it indicates tubular impairment the effect could be a summation phenomenon reflecting the inactivity of damaged tubules and the activity of normal ones.

Excretion of para-aminohippuric acid

The maximum tubular capacity to excrete P.A.H. is decreased in the oliguric and early diuretic phases of oliguric failure (Brun 1954 Ladd 1955) This effect

could be explained either by equal impairment of all tubules or by the combined effect of two populations of nephrons one damaged and unable to excrete P.A.H. and the other capable of normal function

Relationship between glomerular and tubular function

In clearance studies glomerular activity per unit of functioning tubular mass is expressed by the fraction which the G.F.R. bears to the maximal clearance of P.A.H. this normally varies between 1 and 2.5. Low values which indicate a relative decrease of filtration among functioning nephrons were found by Ladd (1955). High values were obtained by Brun (1954) during the early period of renal recovery which suggested that glomerular filtration increased before there was an improvement in tubular excretory power of P.A.H.

PATHOGENESIS

A full understanding of acute renal failure must explain the origin and persistence of renal ischaemia, the probable disproportionate fall in glomerular filtration, proximal and distal tubular necrosis, glomerular lipid changes, the relationship of these to one another and to aetiological factors like oligaemia, fall in cardiac output and release of haem pigments. A schematic outline of the author's concept is shown in Fig. 19. The explanation must include the mechanisms of the oliguric forms of uraemia, the origin of hyperkalaemia, hypocalcaemia and acidosis and the various aspects of tubular function. A full explanation is not yet possible but renal ischaemia and low glomerular filtration have emerged as the central features.

Renal ischaemia and low glomerular filtration

Renal ischaemia as we have seen is initiated by renal vasoconstriction induced by oligaemia and a fall in cardiac output, but the manner by which it is maintained is uncertain. Among the possible causes are humoral vasoconstrictive agents, such as noradrenaline, rennin and 5-hydroxytryptamine and a rise of intrarenal tension. The tense capsule and swollen appearance of the kidney together with the interstitial inflammatory exudate suggest that the intrarenal tension is raised. On the other hand, normal tension was found in dogs with tubular necrosis produced by renal artery ligation (de Wardener 1955) and in patients with acute renal failure (Munck, 1958). The cause of the persistent ischaemia warrants full investigation.

Renal ischaemia presumably initiates the profound fall in glomerular filtration and to some extent maintains it, but changes within the glomeruli may play a part in the latter process. This aspect of the problem also requires further investigation and the finding of multiple lipid droplets in the glomerular epithelium of burned patients suggests that subtle changes opposing filtration may have occurred.

Role of haem compounds

Among the numerous endogenous nephrotoxic agents postulated only haemoglobin and myohaemoglobin seem to be of pathogenic importance. Although

PATHOGENESIS

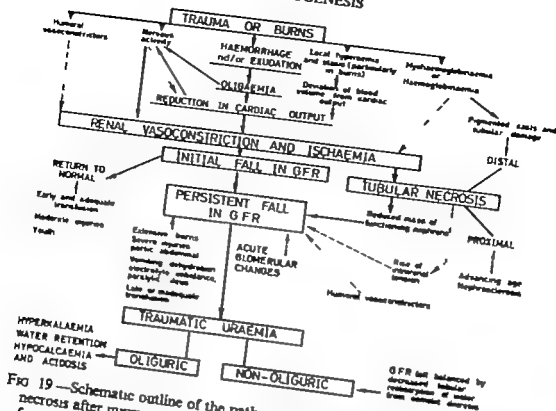


FIG 19—Schematic outline of the pathogenesis of acute renal failure and tubular necrosis after injury and burns. The bold arrows refer to known mechanisms or factors and the broken arrows to suspected or possible ones. (By courtesy of the Editor of the Journal of Clinical Pathology)

it has been claimed that injection of lysed red cells or distilled water can produce severe oliguria and uraemia in normal animals (Reid, 1929 Mason and Mann 1931 Barac, 1952) haem pigments are now considered to act as subsidiary and potentiating factors in acute renal failure. There is a close statistical relationship between haemoglobinuria and acute renal failure after burning, particularly among those patients with diffuse tubular necrosis (Sevitt, 1956 a and b). Release of pigment may turn the scales of a subclinical renal effect or worsen the renal result of dehydration and shock. Thus intravenous injection of lysed red cells produces little change in glomerular filtration in the dog unless the dog is previously dehydrated (Maluf 1949) haemoglobin precipitates fatal uraemia only in animals previously subjected to a short period of renal ischaemia or given a nephrotoxic drug like sodium tartrate (Yuile, Gold and Hinds 1945 Badenoch and Darmady 1948) whilst myohaemoglobin produces uraemia in rats only when dehydration is produced and the legs are crushed (Corcoran and Page 1945).

From the morphological viewpoint, necrosis and rupture of tubular epithelium subjected to ischaemic hypoxia may be facilitated by blockage and pressure of casts. Azotaemia and distal tubular damage can sometimes be reduced or prevented in burned children with severe haemoglobinuria if the blood volume is continuously maintained pigmented casts in the kidney become few as if they are washed out by the flow of tubular urine (see Sevitt, 1957). Renal ischaemia and the deposition of many casts both appear to be necessary for most cases of diffuse distal tubular necrosis after burning.

Summary of evidence against the tubular hypothesis of traumatic uraemia

The concept that the pathological basis of traumatic uraemia is tubular damage or necrosis, that oliguria and uraemia are related to back-diffusion of the filtrate through damaged tubules and that diuresis and recovery are related to regenerating tubular cells is not acceptable for the following reasons

(1) Uraemia without oliguria cannot be explained by non-specific tubular reabsorption or blockage by casts (2) Neither severe oliguria nor the onset of post-oliguric diuresis are adequately accounted for by tubular damage and repair (3) Tubular necrosis has been found to be more common than renal failure in a series of patients at risk (4) Traumatic uraemia may be found without tubular necrosis (5) The electrolyte composition of the urine and the absence of glycosuria indicate continued activity of proximal and distal tubules even though the patient is found to have extensive tubular damage. (6) The pattern of electrolyte excretion can be explained by the hormonal reaction of the body but not by impairment of tubular function (7) Hyperkalaemia, hypocalcaemia, hyperphosphataemia and acidosis result from severe oliguria and do not occur in non-oliguric renal failure even though tubular necrosis is found

Glomerular concept of traumatic uraemia

The concept that renal failure is precipitated and maintained by a low glomerular filtration rate is more fitting to the facts. A persistent reduction of filtration is the basic functional defect and adequately accounts for the azotaemia. The common association of renal failure and tubular necrosis results from common pathogenetic factors particularly renal ischaemia. The development of uraemia without tubular necrosis and *vice versa* reflects other influences at work these include release of haemoglobin or myohaemoglobin which may enhance the likelihood of distal tubular necrosis possible differences in tubulo-epithelial sensitivity to ischaemic hypoxia possible vasoconstrictive humoral agents and subtle changes within glomeruli opposing filtration of which fatty droplets may be a visible sign.

Tubular necrosis never involves all the nephrons the proportion affected varies considerably and affected kidneys have two populations of nephrons, damaged and functioning units.

The damaged nephrons form little or no urine possibly because of back-diffusion of the filtrate or blockage by casts, but they reduce the number of functioning nephrons and add to the diminished rate of glomerular filtration. The filtration rate is, however lower than can be accounted for in this way and indeed tubular necrosis is sometimes absent Damage to a proportion of the tubules may well be responsible for the diminished excretory and reabsorptive powers of the kidneys revealed by maximal loading with para aminohippuric acid and glucose respectively but these are summation effects combining the ineffectiveness of damaged nephrons and the activity of the normal ones

The functioning nephrons are responsible for the formation of the urine, its electrolyte composition, and the reabsorption of the glucose their tubules are capable of reabsorbing the filtration loads of glucose, sodium and chloride and of excreting considerable concentrations of potassium, thereby responding to the demands of increased aldosterone secretion but the filtration load of solutes and water is reduced.

Oliguria is adequately explained by the low rate of glomerular filtration, and other explanations offered such as back-diffusion or blockage of the filtrate by tubular casts are unnecessary. The rate of urine flow is determined by the balance between the reduced glomerular filtration and the reduced tubular reabsorption of water. This is decreased even when oliguria is severe. Oliguria occurs when the flow of urine is inadequate to balance the fall in filtration, but when the absorption of water may be interpreted teleologically as a compensatory attempt to maintain the water balance of the body in the face of a low rate of glomerular filtration and is reminiscent of a similar phenomenon in chronic renal failure (Platt, 1951). When the attempt fails severe oliguria occurs with all its limitations in the excretion of potassium, hydrogen ions and phosphate. In this way the persistent fall in glomerular filtration can be responsible for water retention, hyperkalaemia, acidosis hyperphosphataemia (and thereby hypocalcaemia) as well as azotaemia.

REFERENCES

REFERENCES

- Alwall, N. (1947). *Acta med. scand.*, 128, 317.
 Anderson, T. R., and Steer, A. (1955). Post-traumatic Renal Insufficiency "Battle Casualties in Korea Vol. 4 p 235. Washington: U.S. Army Medical Service.
 Anthonisen, P., Brun, C., Crode, C., Lassen, N. A., Munck, O. and Thomsen, A. C. (1956). *Lancet* 2, 1277.
 Badenoch, A. W., and Darmady, E. M. (1948). *Brit. J. exper. Path.*, 29, 215.
 Barac, G. (1952). In 6th Congr. Int. Path. Comp., 357.
 Brun, C. (1954). *Acta Anesth. Copenhagen*. Munksgaard.
 Bull, G. M., and Dible, J. H. (1953). *Lancet* 1, 603.
 G. Hadfield. London: Churchill.
 — Jockes, A. M., and Lowe, K. G. (1949). *Lancet* 2, 229.
 — — — (1950). *Clin. Sci.*, 9, 379.
 Burnett, C. H. S. L., Shapiro, F. A., Simeone, H. K., Beecher, T. B., Mallory, T. B., and Sullivan, E. R. (1947). *Surgery*, 22, 856.
 Waters, E. G. L., and Bonill, D. (1941). *Brit. med. J.* 1, 427.
 — — — (1945). *Text Rep Biol Med.*, 3, 528.
 — — — (1947). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1948). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1949). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1950). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1951). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1952). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1953). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1954). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1955). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1956). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1957). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1958). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1959). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1960). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1961). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1962). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1963). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1964). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1965). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1966). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1967). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1968). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1969). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1970). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1971). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1972). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1973). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1974). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1975). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1976). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1977). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1978). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1979). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1980). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1981). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1982). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1983). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1984). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1985). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1986). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1987). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1988). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1989). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1990). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1991). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1992). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1993). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1994). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1995). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1996). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1997). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1998). *Proc. R. Soc. Med.*, 50, 498.
 — — — (1999). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2000). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2001). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2002). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2003). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2004). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2005). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2006). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2007). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2008). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2009). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2010). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2011). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2012). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2013). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2014). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2015). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2016). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2017). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2018). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2019). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2020). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2021). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2022). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2023). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2024). *Proc. R. Soc. Med.*, 50, 498.
 — — — (2025). *Proc. R. Soc. Med.*, 50, 498.

TRAUMATIC URAEMIA

- McCracken, B H and Parsons, F M. (1958) *Lancet* 2, 885
- Merrill, J P (1955a). *Milit Med* 117 233
- (1955b). *Treatment of Renal Failure* New York Grune and Stratton.
- Minami, S (1923) *Virchows Arch.*, 245 247
- Muirhead E. E., and Stirman, J A. (1952) *Surgery* 32, 43
- Munck, O (1958) *Renal Circulation in Acute Renal Failure* Oxford Blackwell.
- Oliver J (1953) *Amer J Med.* 15 535
- MacDowell M., and Tracy A (1951) *J clin Invest* 30 1305
- Pease D C. (1955) *J Histochem. Cytochem.*, 3 295
- Phillips, R. A. and Hamilton, P B (1948) *Amer J Physiol* 152, 517 523
- Platt, R. (1950) *Clin Sci* 9 367
- (1951) *Lancet* 1 1239
- Reid, W L. (1929) *Amer J Physiol* 90 168
- Richards, A W (1929). *Trans Ass Amer Physicians* 44 64
- Sevitt, S (1956a) *J clin Path* 9 12.
- (1956b) *Ibid* 9 279
- (1957). *Burns Pathology and Therapeutic Applications* London Butterworth.
- (1959) *Lancet* 2, 135
- Sirota, J H (1949) *J clin Invest* 28 1412.
- Van Slyke, D D Phillips, R A., Hamilton, P B Archibald, R. M Dole, V P., and Emerson, K (1944) *Trans Ass Amer Physicians* 58 119
- Ewan, R. C., and Merrill, J P (1953). *Medicine Baltimore* 32, 215
- Taylor W H (1957) *Lancet* 2, 703
- and Reid, J V D (1956) *J clin Path* 9 184
- Teschan, P E., Post, R. S., Smith, L. H Abernathy R. S., Davis, J H., Grey D M., Howard, J M., Johnson, K. E., Klopp, E Mundy R. L., O Meara, M P and Rush, B F (1955) *Amer J Med.* 18, 172.
- Trueta, J., Barclay A E., Daniel, P Franklin, K J., and Pritchard, M M L. (1947) *Studies of the Renal Circulation* Oxford Blackwell.
- de Wardener H E. (1955). *Lancet* 1 580
- Yuile, C L., Gold, M A., and Hinds, E. G (1945) *J exp Med.*, 82, 361
- Zollinger H. U (1952) *Anuric bei Chromoproteinaemia* Stuttgart Thieme.

PRINCIPLES AND TECHNIQUE OF WOUND SURGERY

CHAPTER 8

RUSCOE CLARKE

INTRODUCTION

ALTHOUGH antibiotics provide no substitute for surgery in the treatment of wounds it is only in the antibiotic age that there has been any wide agreement on the details of wound treatment. This is less definite where primary measures have failed to prevent infection or breakdown. Only some of the principles of modern wound treatment can be supported by experimental evidence, others are basically empirical but they work. The approach of several converging trends is essentially that of Halsted (1924) that uninjured tissues can best resist infection. The object of surgery is to preserve tissue vitality so that bacterial contaminants are not provided with culture media from bruised, damaged or devitalized tissues, from blood clot or serum or through interference with the blood supply by swelling in a closed compartment. The principles which Halsted derived from animal experiments and Essex Lopresti (1950) summarized evidence that the frequency of primary bacterial contamination of wounds is low. Moreover most wounds can be cleaned in the early stages so that viable tissues can complete the process of sterilization whatever the degree of primary contamination. Antibiotics can destroy or inhibit bacteria without destroying the tissues, thus differing from most other antiseptics. They are not effective when organisms are protected by barriers of bloodless material whether injured tissues or foreign matter introduced at the time of wounding. The aim of treatment is to leave the tissues capable of destroying any bacteria left after cleaning it is necessary occasionally to reinforce this with courses of selected antibiotics. These should be prescribed infrequently to minimize the development of resistant strains, particularly of *Staphylococcus aureus* (Lowbury 1958).

Just as survivors of the Listerian age like the ritual anointment of the appendix stump with carbolic acid linger on, so useless gestures of *in vivo* antiseptics some times colour the treatment of accidental wounds. It is time that their theoretical pointlessness should be widely taught. We are reminded of Wright's (1942) polemical battle with the giants of surgery and of the sleepless night of Paré (1545) who had run out of boiling oil and was unable to burn the wounds of his patients—only to discover that it wasn't really necessary.

The antibiotics have given us a second string to our bow so that spreading streptococcal infection is now almost a thing of the past. They have given us confidence to do things which should have been done before and which can be done now with a greater chance of success. We must not forget what was achieved without them by surgeons who thought in terms of biological reactions of the soil as well as the seed of the reinforcement of natural powers of resistance rather than in crude terms of wound sterilization.

The primary objective of treatment is the prevention of clinical infection so that healing can proceed uneventfully. Even the control of haemorrhage is usually incidental, although occasionally overriding in importance. The prevention of infection removes the chief danger of secondary haemorrhage, which in civilian surgery should be little more than a memory. It paves the way for planned primary repair of divided tissues and the replacement of missing tissues by grafts or prostheses. The elimination of infection reduces scar tissue, accelerates the speed of union, allows early rehabilitation and return to normal function.

Leriche's (1939) aphorism that the best antiseptic is the knife still holds good, but terminological arguments sometimes obscure the role the knife must play. Wound surgery was once conceived as involving radical excision of the wound either *en bloc* with the contaminating bacteria still adhering or layer by layer (with a fresh knife for each) so that all tissue exposed to contamination was cut away. The first procedure is possible in some superficial wounds whilst the second has a partial value in others, but the conception is wrong because it is impossible or unnecessary in most serious wounds. The concept of surgical removal of all contaminated tissues which the word excision implies should be abandoned. Highly-contaminated battle injuries are least suitable for its application whilst it can do much harm in certain civilian injuries such as open fractures of the leg. Even its protagonists make exceptions for the hands and face where unnecessary removal of tissue is so obviously harmful.

The term *débridement* which never meant excision as some essayists once maintained, cannot be revived and it is too late to reintroduce its original meaning of wound enlargement. The term "wound toilet" is descriptive although it does not convey Leriche's honour to the scalpel. It does seem a pity that while we look for substitutes for the umbrella word "shock" we are left without a term to convey what surgeons should do for the wound.

WOUND CONTAMINATION

The degree of primary contamination depends on the causative instrument, the circumstances of wounding, and other factors. Perhaps as important as the number of pathogenic organisms is the manner in which they are distributed and the extent to which they are protected by foreign matter. The nature of the dirt matters at least as much as the number and nature of the germs. The wound made by a fracture from within outwards may have minimal contamination whilst the wide open wound sustained in a muddy road will be very dirty but relatively easy to clean. The deep penetrating wound with dirty clothing driven ahead of the entering object can be the most dangerous combination of contaminated dirt, damage to the blood supply of muscle in which protected germs may lodge and thrive, and difficulty of access. In contrast the majority of light-engineering industrial wounds are nearly sterile on infliction whilst burns are sterilized by the act of burning.

Much of the primary contamination of wounds is derived from the clothes and skin of the patient. This became important during World War II when surgeons transferred their attentions from the Libyan desert to the cultivated terrain of Italy (Cope 1953). In the desert the techniques of wound exploration based on World War I experience appeared no longer necessary and this was unfortunately attributed to the use of sulphonamides. Experience in Italy showed this to be an

THE MINOR WOUND

Illusion full wound exploration and decompression became essential. The difference depended not only on the relatively germ free desert but also on the fact that the wounded men were often wearing little more than a thin pair of shorts. A dangerous feature in Italy was often the germ-laden clothing in the depths of the wound.

Foreign bodies

Penetrating foreign bodies may require urgent removal but we must be clear about priorities. Their removal should mean that the wound has been fully explored and that organic material present has been removed. Metallic foreign bodies are the least harmful from the point of view of infection. They are clearly visible radiologically but least important as a focus of infection and their removal may be a luxury in difficult cases. Many a wound has been adequately explored and treated without the foreign body being found.

Secondary contamination

The value of first-aid treatment in the prevention of infection depends on the relative lack of primary contamination compared with the danger from organisms subsequently introduced by the patient or others (De Waal, 1943). Correct first aid treatment involves covering the wound or burn with a suitable dressing and leaving it covered until medical authority decides further. A sterile gauze dressing or lint, a recently washed handkerchief, towel or piece of linen, or failing these the cleanest piece of material available should be bandaged on and left. If there is bleeding a pressure bandage should be added. When the patient reaches medical care the doctor or nurse should wear a mask, wash and then dry the hands, remove the old dressing with forceps, examine the wound visually and decide on further treatment. This routine should be possible at every hospital, clinic, health centre or surgery. If treatment cannot be completed immediately a sterile dressing is applied with forceps and the wound rebandaged. If this can be postponed until the doctor who is going to operate is available so much the better; if not, the wound has to be exposed for another few seconds. The risk of secondary contamination from hands, clothing, noses and throats is thus kept to a minimum. If the above procedure is omitted nothing vital has been lost provided that nothing which may be dirty touches the wound and that the latter is only exposed for a few seconds. No cleaning is done at this stage unless the wound is trivial and treatment is to be completed forthwith.

THE MINOR WOUND

Trivial and superficial wounds should be cleaned with soap and water preferably under a tap with running warm water. They may then be dressed with dry gauze and a bandage, left open to the air or protected by a drying agent such as Nobectane which acts as a minute dressing or artificial scab. No wound is so small or so clean that it is not best cleaned by soap and water or a mild detergent solution; no antiseptics are necessary and most of them are harmful to cells as well as bacteria. Even the antibiotics in excessive concentration can inhibit cell growth (Cruickshank and Lowbury 1952; Funan Hu and his colleagues 1956).

THE MAJOR WOUND

The local treatment of major wounds can be summarized in the following stages.

- (1) First-aid cover dressings.
- (2) Diagnosis.
- (3) Theatre cleaning exposure, exploration, and more cleaning removal of foreign matter essential removal of dead muscle removal of other non-viable tissue as indicated decompression suture or grafting dressing and bandaging plaster or other splintage.
- (4) Elevation.
- (5) Planned review of the dressing after a given time or as indicated.
- (6) Observation, local and general.
- (7) Chemotherapy

A wound requiring exploration or repair needs treatment in an operating theatre cleaning is then deferred.

The wound having been inspected further information may be necessary for example about bones by radiological examination, about nerves and tendons by clinical testing. Other injuries must be investigated and the patient's point of view considered. In wounds of the extremities where either amputation may be needed or plastic procedures are possible the problem should be discussed with the patient or relatives. If haemorrhage has been appreciable or if surgery may lead to further bleeding, transfusion should be organized. Bleeding wounds may require early surgery since a steady ooze through bandages can become serious.

Use of tourniquet

Haemostasis is desirable and the availability of bank blood must not encourage the attitude that bleeding does not matter. Nevertheless its availability may help to decide whether surgical treatment of an extremity will be done under a tourniquet. The author's preference is to avoid the use of a tourniquet for the following reasons: bleeding vessels can be controlled immediately and the nature of any vascular damage therefore becomes clear at an early stage; the viability of skin or muscle can be better judged from colour appearances and bleeding on incision; nothing further is done to reduce even temporarily the blood supply to injured tissues; and the risk of damaging brittle arteriosclerotic arteries is avoided.

A tourniquet applied pre-operatively may be left on during cleaning. With experience more blood is seldom lost without a tourniquet than with one. Wide exploration often requires no more than skin incision in contrast to the dissection of tissue planes needed for the deliberate procedures of cold surgery.

Anaesthesia

Few significant wounds can be treated properly without anaesthesia. Many surface abrasions with grit or gravel rubbed into the wound give the best cosmetic results when scrubbed under anaesthesia. In Great Britain there is a high standard of general anaesthesia and a low mortality from anaesthetics for minor operations. The biggest risk is inhalation of vomit and the anaesthetist should be given every chance to take full precautions. There are however many minor wounds which can be effectively treated under local or regional anaesthesia, particularly brachial

plexus block although even this carries the risk of an occasional pneumothorax. Wound surgery is possible under local anaesthesia for wounds of any size involving the trunk or lower limbs but requires considerable experience so that the full potential operation field is considered from the start. General anaesthesia is preferred in Great Britain for major wounds and certainly simplifies the surgeon's task.

Cleaning

The wound should be cleaned by the surgeon first because this is often the most important part of the operation and secondly because, during the cleaning further information may be gained about the nature and extent of the damage. Adequate quantities of soap and water or soap or detergent solution, remove microscopic dirt and aim at social cleaning rather than sterilization. This removes the dirt which shelters bacteria from attack by the body defences. The water is as important as the soap and the procedure is quite different from the ritual wiping that is often substituted for it. The skin widely around the wound and near any part of the possible operation site is cleaned in the same way as one would clean the dirty knees of a child who had fallen in the mud. The author has found no harm come from including the wound in the skin-cleaning process. The same process may be continued after the wound is opened, although many surgeons prefer to wash inside the wound with water or saline. A thorough washing may take 15-20 minutes or longer. The area is then dried, towelled off, and is ready for the knife.

Exploration

The role of surgery is not excision of the wound. It involves first the wide opening up of the wound to expose the full extent of the damage, secondly the excision of certain damaged tissues, and thirdly wound repair. Surface wounds and civilian injuries caused by industrial rollers or large bus tyres may already be wide open. Most other major wounds require extension or an independent incision to expose contamination with dirt and foreign material, the extent of damaged tissue and the nature of the internal repair required. Generally wound extension or separate exposure requires incision through skin and subcutaneous tissue only.

The extent of exposure depends on the nature of the wound, the likelihood of deep spread of foreign material, and the possibility of damage to important deep structures. It may be limited by the possible harmful effects of extensive skin incision. The first two considerations depend on the mechanism of injury, the latter two on its anatomical site. Battle wounds, explosive wounds and injuries due to high velocity missiles often have deep damage out of proportion to the surface wound. Small penetrating bullet wounds may traverse important structures. Jagged metal fragments large or small, may damage muscle out of proportion to the surface wound. A feature of high-speed missiles is that by striking bone they give rise to secondary missiles. In civilian trauma similar injuries may arise from explosions, including those from domestic boilers and home made bombs.

The deep damage in the crushing, tearing and twisting injuries of civilian life differs from that of war missiles particularly when fractures are compounded from within. This is perhaps fortunate, for in those patients with extensive undermining of skin and subcutaneous tissue wide extension or new incisions may interfere with

skin blood supply. The muscles are split rather than burst and unless there is concomitant damage to muscle blood supply the risk of gas gangrene is less than with war wounds. When gas gangrene has occurred nothing short of full exploration will suffice. Other exceptions to the need for wide exploration are certain penetrating and perforating wounds in which full exploration would risk damage or spread of infection to deep structures without possible benefit. This refers particularly to wounds of the hands and feet from nails, and to through-and-through wounds of the limbs or chest then it is usually sufficient to deal with the wounds of entry and exit without laying open the whole limb dividing major muscles transversely or doing a thoracotomy. By contrast similar wounds of the head or abdomen should rarely escape exploration of the affected cavities.

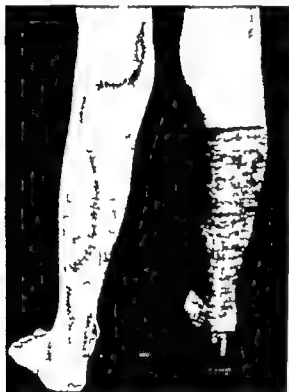


FIG. 20—A harmless incision for exploring the calf and popliteal fossa.

Skin Incisions

Knowledge of the fate of incisions in particular sites and directions has led to modification of those used for wound exploration. Straight incisions are best replaced by winding incisions resembling an S or double S and must never run vertically on the front or back of joints, for this can lead to crippling contracture. The vertical winding incisions advisable for leg and thigh and arm and forearm can be joined by horizontal incisions in the joint creases of the knee, elbow and wrist (Fig. 20). Alternatively the joint is crossed laterally or obliquely. Existing wounds can either be enlarged so as to convert them into a double flap or ignored and left in the middle of a flap for independent repair (Fig. 21). When skin and subcutaneous tissue is missing the planning of incisions and the subsequent closure of defects is aided by the principles of plastic surgery in relation to the placing of incisions. The enlargement for exploration must be designed to facilitate

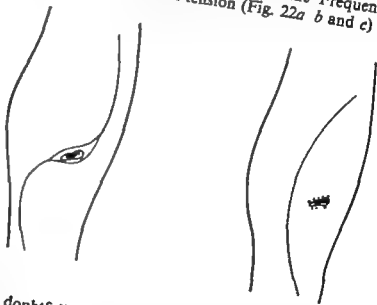
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closure and leave suture lines and grafted areas suitably placed away from skull defects, plated bones or other sites where breakdown would be more than a minor complication. Formerly the tendency was to explore first and repair afterwards. It is now possible with experience to anticipate the deep problem and to plan the original skin incision with closure already in mind.

It used to be taught that wound enlargement should be preceded by excision of skin margins but during World War II excessive skin excision was often found to do much harm. Taking away a quarter of an inch from each edge can cause pressure tension after closure and lead to skin necrosis and a bigger skin defect. Split-skin grafting is no longer considered a matter for the plastic expert and many wounds can be grafted easily without risking tension although in the majority repair with whole skin is preferable. Skin excisions may be deferred until exploration has revealed the extent of the problem and the repair required. Then it may be advantageous either to excise rather more skin or to excise none at all beyond the obviously useless.

In any case skin excision may need to be minimal particularly when the face or hands are involved and where it may lead to tension on the suture line. Frequently preservation of all the skin allows suture at normal tension (Fig. 22a b and c).

FIG 21—Alternative incisions for wound exploration over a joint.



Excision

Excision of all non viable or doubtfully viable tissue is no longer the rule. Modern practice requires independent consideration of each tissue first its possible role in predisposing to infection and secondly in relation to repair even if it is only to be used as a graft.

When the wound is opened it is again cleaned to remove dirt, foreign material and foreign bodies but deeply placed metallic bodies may be left if they are difficult to find provided that every branch of the wound track has been explored and decompressed. It is then possible to see to what extent important deep structures such as vessels or nerves are involved and to secure haemostasis with artery forceps and non irritant ligatures.

Surgery was inevitably crude in the improvised surroundings available for treating mass casualties during wartime. Excision was carried out piecemeal and repair was left until later. It is now clear that only muscle should be extensively

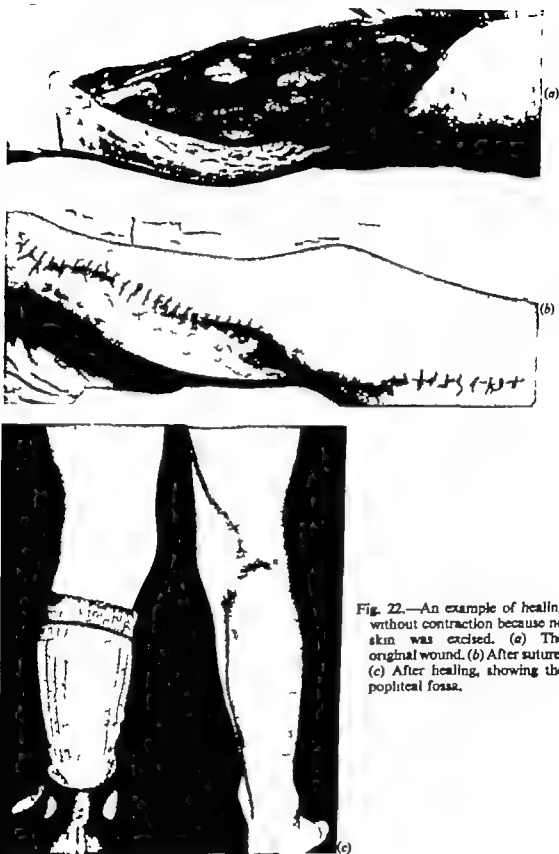


Fig. 22.—An example of healing without contraction because no skin was excised. (a) The original wound. (b) After suture (c) After healing, showing the popliteal fossa.

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resected, because dead and damaged muscle predisposes to gas gangrene even with prophylactic antibiotic therapy. With better facilities and more time and experience in the different types of civilian injury even muscle can be excised with discrimination. Small amounts have often been left without causing gas gangrene and the full value of the antibiotics in prophylaxis is not known. Any sizeable mass of dead muscle which does not contract when pinched or bleed when cut must be removed. If this can be done to leave a "saucerized" wound or one that will fall together with skin suture so much the better. Segments of transected and devitalized muscle should be "scrapped". Bruised muscle may be discoloured but viable sub-fascial ecchymosis can make a muscle look black although it is seen to be normal when incised. After some hours the onset of gas gangrene may be indicated by the peculiar brick red or salmon colour of the muscle turning to green grey and dirty brown, whilst simultaneously a characteristic odour often develops. In most civilian injuries devascularized muscle is excised but when viability is doubtful small muscle masses may be left if they are likely to be useful either as muscle or in filling up a dead space which would otherwise permit the accumulation of blood.

For all other tissues the need to excise depends on specific circumstances. Connective tissues do not readily predispose to infection even when left deprived of their blood supply. Free autografts of bone, cartilage, tendon, fascia and dermis are all in common use and can be incorporated in the body. Separated and damaged fatty tissue is liable to undergo liquefactive necrosis and is best removed. Injured tendons left in a wound may slowly die and require subsequent resection or they may slowly become revascularized. Damaged tendons should be excised if they are likely to be useless and particularly if by adhesion they may cripple the action of attached and functioning neighbouring tendons. Occasionally non-functioning tendons provide a useful tenodesis. Fascia which is partially disconnected may be preserved for covering bone or an open joint beneath a skin defect. Cartilage over bone and partly attached ligaments will survive if covered with whole thickness skin and subcutaneous tissue.

Bone is the most important connective tissue to preserve. Before World War II it was taught that all bone deprived of its vascular attachments must be removed because it predisposed to infection, but it is now known that this is not so even when the bone is completely separated. It will die but it is often subsequently revascularized, incorporated and reconstituted by new living bone although this process may take months or years to complete. If infection does not start elsewhere the presence of dead bone will not make it more likely. It was therefore proposed to establish the principle of "grafting the bone defect with any available loose fragments" (Cope, 1953). Such fragments are most useful for filling up defects in long bones and when they are not available the gap may need to be bridged by a newly cut bone graft (Fig. 23). Bone defects in severe open fractures have been successfully grafted with large bone fragments which have fallen on the floor and been replaced after boiling.

Decompression

A major predisposing cause of severe infection is deep tension complicating penetrating wounds, especially war wounds of enclosed regions like the calf or anterior compartment of the thigh. This can lead rapidly to fulminating infection with septicaemia and systemic deterioration even without the development of

typical gas infection. The wound and the deep fascia enclosing the compartment should be opened widely. For example, in the thigh a longitudinal incision through the fascia lata may allow divided tissue to fall together. Subsequent tension can only be prevented by the addition of transverse cuts. The fascia must be incised one way or another until there is no tendency for muscle to bulge and then a little more to allow for further swelling. This decompression will often expose deep blood clots and allow access to damaged vessels. It is most urgent in battle injuries but equally necessary in many deep civilian wounds, especially severe fractures of the leg with small skin openings. In the past decompression was sometimes followed by drainage. With early operation drainage is unnecessary and it



FIG. 23—Radiographs showing a gap in the tibia grafted originally from the same leg. (a) The graft united. (b) After reinforcement with a further graft from the other leg.

leaves a pathway from the surface whereby secondary infection may occur. The swelling of deep muscles which made decompression necessary does not preclude immediate skin suture or grafting.

Haematomas and bandaging

Deep collection of blood is a major factor predisposing to wound infection as it is an excellent culture medium for some organisms. Accurate haemostasis by clipping bleeding points and ligation with fine non-absorbent sutures may control obvious bleeding but it will not prevent further oozing. Control of deep oozing depends on efficient bandaging which should be carried out by the surgeon. In addition to a capacity to absorb blood and discharge the dressing materials used must possess some elasticity which allows even bandaging at the right pressure to

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control deep oozing without producing strangulation or venous obstruction. Dressings and cotton wool must extend well beyond the wound and up and down the limb because tissue planes are opened up at a distance from the wound. The bandage shown in Fig. 24 was adequate for the skin wound but gave little support to the tissues into which extravasation could occur.

With small wounds suture of the skin often allows its fibro-elastic qualities to provide the necessary pressure. Pressure dressings are necessary for large wounds particularly when skin is undermined, dissected or only loosely attached, because there may be much room for swelling to take place. Before the wound is closed it is necessary to envisage its state after the dressings have been applied, particularly the possibility of spaces which may not be obliterated by suture. When dead spaces are inevitable they may be drained and the drainage tubes used for local instillations of antibiotics but this should rarely be necessary.



FIG. 24—An open fracture of the femur showing the inadequate bandage which probably contributed to infection. It should have included the whole thigh and knee.

Skin closure

The wound has now been widely opened and explored. dirt and foreign bodies have been removed. dead muscle and perhaps some other devitalized tissue have been excised. Consideration has been given to repair of deep tissues of bone by nailing, screwing, wiring or plating of main vessels by suture or grafting of tendons occasionally of main nerves. The wound has been decompressed. Open joints have been washed out and closed. The wound has been irrigated in all its corners. There remains the problem of the skin.

It is necessary to correct some misunderstandings. Many patients have died whilst surgeons learnt the hard way certain "do's and don'ts" of the treatment of war injuries. These injuries must be widely opened up to expose the extent of

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injury and remove dead muscle but they must never be sutured if the patient is going to be moved or if facilities for careful supervision are not available. This applies essentially to limb wounds and many trunk wounds where movement will inevitably provoke a haematoma which if tightly enclosed will go septic. It does not necessarily apply to wounds of the hands and face where other considerations make early closure desirable. Other factors determine why patients with abdominal injuries do not travel well. Drainage tubes need not be used because the skin is left open and the opening of the wound must not be nullified by dressings which act as a plug. All that is necessary is a strip of gauze or Tulle Gras dressing in the wound and bandaging as described above. If all goes well the wound can be treated by delayed suture—that is, suturing the wound as a second-stage procedure. It will often be sterile and can be sutured easily and reliably provided that the dressings have not been disturbed. If they have been removed for inspection more than once surface infection is likely and delayed suture less successful.



FIG. 25—A wound that was cleaned and sutured. (a) The original wound. (b) After healing. (A small superficial slough was excised and grafted.)

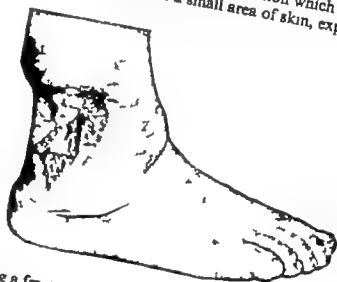
Delayed primary suture is rarely needed in civilian surgery except in an occasional gravely injured patient with a limb needing amputation through skin of doubtful viability. It is important for the less experienced surgeon to know that a wound can be left wide open in an ill patient or when the wound is complex it can be dressed and suture postponed for 4–10 days.

In most civilian patients careful wound surgery will be followed by primary suture or grafting (Fig. 25). Primary suture should be the rule when there is no loss of skin and it is often the treatment of choice even when the wound appears tense and suturing difficult. The thing to do is to start sewing and see what happens. If the skin is stretched to the point of going white or looking shiny the procedure must be abandoned. Suture at normal tension is beneficial to the skin and makes for a normal pattern of vascularity. During direct stitching the skin should not "tent" over dead space left by excision of deeper structures or their separation. Deep sutures are not required when the deep structures are approximated by skin suturing. Occasionally dead space can be avoided by strategically placed deep stitches. Suturing is sometimes advisable to cover bones or open

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joints with a layer of fascia, aponeurosis or sometimes muscle. In general however the less deep suturing the better. Tenting from small excess of skin may be overcome by gently pushing the excess into the wound depression with wet cotton wool. Superficial dead spaces should always be avoided because of the risks of infection in the clot and of sloughing of unsupported tissues over them. When suture is possible drainage is rarely indicated. Successful primary suture converts an open wound into a closed one with normal skin providing the best barrier to bacterial invasion. It provides the best cover for primary repair of deep structures and allows early delayed repair of injuries to major nerves when this is indicated. Success depends on avoiding deep infection and on the survival of the skin. The first risk may be reduced by the use of antibiotics but failure is to be regarded as a failure of technique. Death of sutured skin is more complex. Necrosis of a narrow band along the sutured margins may lead only to a small granulating area this will either heal rapidly or require minor grafting or perhaps secondary suture. However on the face or hand it may produce an imperfection which would not matter elsewhere. More serious is breakdown of a small area of skin, exposing

FIG. 26.—Result of primary split-skin grafting for a deep hole near the ankle.



an open joint or a metal plate fixing a fracture. This might be avoided by planning the original incision to avoid crossing points of danger particularly when the skin is of doubtful viability. A large area of skin slough over muscle or fascia (away from joints where scarring can cripple) can be excised later and replaced by a split-skin graft. When limited skin death is probable skin should be excised primarily and replaced by a graft usually of split skin when the affected area lies over a viable soft tissue base. The hand face and scalp most often require full thickness skin replacement rather than thin surface grafts. A knowledge of the whole gamut of procedures for skin replacement and transfer is useful but in acute injuries it is wise to use the simplest that will suffice. Pedicle rotation flaps, advancement and relieving incisions assume the presence of normal skin and subcutaneous tissues but in acute injuries with sizeable skin loss the surrounding skin may be damaged although it looks normal at the time. Risks should not be taken unnecessarily. Simple grafting is sufficient when skin loss is moderate or when removal of dying skin leaves a viable area. A complex cavity may be lined with grafted skin packed gently to exert an even pressure against the walls (Fig. 26). Grafting over holes

with possible contamination is not dangerous—any discharge which forms will push the graft away and graft failure is all that will happen. These split skin grafts should be applied if possible in a single piece bigger than the area to be covered even after tucking down to line the depression. The overlap allows for possible skid during dressing. The graft can be anchored by Tulle Gras dressing and careful bandaging. A few stitches may be necessary but detailed suturing should be omitted. A bigger problem is presented by certain run-over injuries in which much of the leg is flayed but deeper structures are intact the skin is still available and some of it may live. The simplest procedure is to clean and replace it all by suture. It is then treated like a grafted area skin sloughs are excised later and replaced with split skin. When there is only partial cover with skin its replacement must be associated with grafting and nothing will have been lost.

The electric dermatome facilitates the taking of skin especially when multiple limb injuries make it difficult to obtain a graft by ordinary methods

Splintage

Major open fractures of long bones require splinting, sometimes from within. All other major limb wounds warrant early immobilization a full plaster cast over dressings is often desirable, both for comfort and because it avoids movement which may disrupt early healing. It also protects the part from being knocked and discourages frequent dressings. Primary application of plaster must not be equated with the closed plaster treatment of Ollier (1872) Orr (1942) and Trueta (1943). Although many properly explored wounds heal under plaster without suture or grafting the introduction of the closed plaster technique for war wounds held up the planning of the two-stage operation of delayed suture and the discussion of the principles of primary suture. Trueta (1943) defined the principles of wound treatment carefully but circumstances made him overplay the role of plaster. Plaster applied primarily can be removed for subsequent wound dressings when applied over the unsutured wound it must come off for early suture long before it begins to smell. The great advance made with the closed plaster treatment was the rejection of the previous practice of daily dressings. Wounds are now dressed as infrequently as possible and usually for definite indications rather than the satisfaction of curiosity. Careful primary treatment permits immediate wound closure or completion of closure at the next dressing. The Winnet Orr technique has a useful but limited place for an occasional patient with a chronic wound infection resulting from an infection of the bone. The modern aim is to secure healing by skin closure or grafting rather than by time and granulations.

Antibiotics

Penicillin transformed the prognosis of many war wounds. It is justifiable to give it to every patient with a contaminated major wound beginning with a large pre-operative loading dose and then full dosage for 5-7 days. The course can often be stopped early as it is often possible to decide that primary infection has been avoided and is unlikely to develop later. Some wounds can be presumed to be contaminated with penicillin-resistant organisms particularly those involving the bowel, near the buttock, or which have been widely exposed to the risk of contamination in hospital. Alternative or additional antibiotics may then be used.

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When infection does develop it is important not to rely on ringing the changes of antibiotic drugs without considering further local treatment (Lowbury 1958)

Review of the dressing

The first post-operative dressing is usually indicated between the fifth and the tenth day earlier when infection or skin necrosis is likely later when a split-skin graft must not be disturbed. Unexpected pain and a rise in temperature after the first 24 hours may indicate infection and the need to inspect the wound although there is rarely need for precipitate action. Rest is still a valuable part of the treatment of early infection.

Immunization

There is no clinical evidence to support the use of anti gas-gangrene serum for the prevention of anaerobic myositis. Its use in treatment is doubtful in these days of antibiotics (Roggers 1945).

Tetanus

Tetanus still kills 50-100 persons each year in England and Wales and can be prevented by immunization with toxoid. This should be much more widely carried out in infants and agricultural workers. Immunization must not only be regarded as a prophylactic measure against tetanus, but also against serum sickness and allergic reactions which may follow the use of antiserum.

In the absence of active immunity antitetanus serum still has an important role in reducing the incidence and severity of the disease. It is probably unwise to suggest that every patient with a surface injury should be passively immunized but tetanus antiserum should be given in the following circumstances: (1) in all wounds contaminated by dirt from road or field (2) in severe injuries and deep penetrating wounds (3) in wounds complicated by infection (4) in any patient where there is some degree of special risk. The danger of developing a sense of false security is illustrated by a patient who died from tetanus following a minor factory injury. He did not receive antitetanus serum and it was not until the inquest that it was learnt that the vehicle on which he had been working had lain out in the fields for many months!

A second dose of antitoxin may however be of little value since it is often eliminated very rapidly (Payling Wright, 1958).

Transfusion

The indications for transfusion are dealt with elsewhere but the following points are emphasized: (1) Adequate primary transfusion allows time for careful wound surgery. (2) Full and early replacement of blood loss opens up the blood supply to injured tissues. (3) Early and adequate transfusion reduces subsequent dietary difficulties. (4) Infection causes anaemia which is best treated by transfusion. (5) For the anaemic patient with a wound infection transfusion is an important part of the treatment of the infection. (6) Stored blood is as good as fresh blood for all these purposes.

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The time factor

In conclusion a word is needed about the time factor. It has not yet been proved that delay demands any radical change in the principles of wound treatment so long as infection is not clinically manifest. Ideally a wound is better dealt with surgically within the first 6 hours but unless there is a mass of damaged muscle predisposing to gas gangrene most wounds can be left if necessary for 24 hours or perhaps longer and then treated in the same way. We do not know how long the delay can be extended by early chemotherapy probably every hour counts to some extent and surgery should be performed as soon as the patient's general condition allows usually within a few hours of injury.

When infection is evident surgery must be either more radical or confined to wide exposure and excision of damaged muscle. Appearances are often deceptive and apart from gas gangrene it is better when in doubt to do little surgically and to test the value of splintage, rest and chemotherapy. Attention to nutritional, fluid, electrolyte or red-cell needs are invaluable.

REFERENCES

- Cope, Z. (1953) *The History of the Second World War Surgery* London H M Stationery Office.
 Cruickshank C. N. D. and Lowbury E. J. L. (1952) *Brit med J* 2, 1070.
 Essex Lopresti, P. (1950) *Lancet* 1 745.
 Funari Hu, Livingood, C. S. and Hildebrand, J. F. (1956) *J Invest Derm.*, 26, 23.
 Halsted W. S. (1924). *Collected Works Surgical Papers of William Halsted* Baltimore Johns Hopkins Press.
 Leriche, R. (1939) *Lancet* 1 803.
 Lowbury E. J. L. (1958). "Chemotherapy in Surgery" *Textbook of British Surgery* Vol. 3 Ed by H. Souttar and J. C. Golligher London Heinemann.
 Ollier L. (1872) *Bull Acad Med., Paris* 1 243.
 Orr H. W. (1942) *Wounds and Fractures*. London Baillière, Tindall and Cox.
 Paré, A. (1545) *Le Méthode de Traiter les Playes Faictes par Harquebudes et Autres Batons à Feu* Paris.
 Payling Wright G. (1958) *Proc R. Soc Med.*, 51 997.
 Rodgers, H. W. (1945). *Proceedings of the Congress of Central Mediterranean Force Army Surgeons, Rome* p 119.
 Trueta J. (1943) *The Principles and Practice of War Surgery* London Hamish Hamilton.
 De Waal, H. L. (1943) *Edinb med J.*, 50 577.
 Wright, A. E. (1942). *Pathology and Treatment of War Wounds*, p 175 London Heinemann.

CHAPTER 9

PRINCIPLES OF THE TREATMENT OF FRACTURES

F G BADGER

EACH injury is unique and should be treated on its merits. Classification may be convenient for teaching and assessing broad lines of treatment, but it usually means that a variety of injuries are grouped together because they occur in a particular anatomical region.

Treatment must depend on diagnosis which requires more than a radiological examination. Because too many doctors think that radiological examination is a safe way of avoiding litigation, radiography is used indiscriminately. At times the immediate taking of radiographs may even be detrimental and constitute negligence in itself. It is only part of the examination and not necessarily the decisive part. Usually only the bone is shown and there is often a tendency to treat the radiograph rather than the patient.

When a bone is fractured the surrounding soft tissues are damaged to a greater or lesser extent. When the fracture is minimal and the soft-tissue injury slight the limb can be used and the lesion may be better regarded as a "bruise". Otherwise the injury should be viewed as the broken limb rather than as a fractured bone.

The intact skin of the closed fracture conceals the extent of the trauma, whilst even in the open fracture only part of the damage can be seen in the wound. At autopsy or surgical operation it is often possible to see just how widespread the damage can be.

The word "fracture" has assumed undue importance and even emotional significance. Emphasis on the injury to one tissue may overshadow the other torn, lacerated or crushed tissues—muscle, tendon, fascia, ligaments, nerves and blood vessels. The bone is clearly shown by radiological examination, but the extent and significance of the soft tissue damage can only be appreciated from a radiograph taken in deformity before reduction or during manipulation. Such films can indicate that certain tissues must have been wounded so that the pattern of associated soft-tissue damage can be deduced.

HISTORY

In addition to careful clinical examination and radiography a detailed history is required from the patient or from someone who actually saw the accident. At times both accounts may be valuable. Careful notes made at the first examination can be particularly useful if any litigation is involved.

The history however may be incomplete or misleading, rarely wilfully so. The old lady with a fracture of the femoral neck usually claims to have fallen on the hip. But her fracture was caused by torsional violence. Once the bone had broken she fell and lying on the floor with a painful hip she assumed that she had fallen.

PRINCIPLES OF THE TREATMENT OF FRACTURES

on it. How long she had been lying before help arrived may be valuable information several hours in the cold immobile on the floor may well have caused the "bedsores" discovered later in hospital

Abrasions or contusions over the greater trochanter are not seen in such patients. When bruising on the outer aspect of the hip is associated with a fracture it is usually the greater trochanter acetabulum or pubic ramus that is involved.

The history of a fall on the outstretched hand is given in many fractures of the upper extremity from scaphoid to scapula.

MECHANISM OF INJURY

Knowledge of the mechanism may be valuable the causative violence may have caused bending, twisting, "telescoping" shearing, crushing, rolling or pulling. Forces may be combined these may be applied in varying rotational positions and may take effect in different positions of joints. The nature of the violence may be valuable in understanding combinations of injuries such as when an associated nerve injury is due to torsion and traction. A second violence may occur for example by a drunk trying to walk on an already broken leg. Continuing violence may alter its nature or direction particularly when lines of force are changed after an initial fracture or dislocation so that distant structures are involved in a complex manner.

Displacement may be due to one or more of the following factors the original violence muscle action, the effects of gravity the misguided efforts of a first-aid worker or inappropriate medical care.

CLINICAL EXAMINATION

A detailed clinical examination is required so that the full extent of the injury can be appreciated. It needs to be painstaking in the unconscious patient in whom an injury may be easily overlooked.

Examination involves inspection and palpation, gentle passive and active movements to test the integrity of specific structures and tests of sensation and vascular sufficiency. Every step must be designed to elucidate anatomical and functional features of the lesion.

RADIOGRAPHY

A solitary film may fail to show a fracture or a displacement, whilst an artefact may be mistaken for a lesion. At least two views at right angles to each other must be taken some injuries need more. From the radiographs an attempt is made to create a three-dimensional mental picture of the broken bone.

From the knowledge gained at autopsy and operation, the fractured bone can then be invested with a picture of the injured soft tissues. Examination under anaesthesia may be required for a full appreciation of the extent of the trauma the mobility of the bony fragments can be felt and further visualized by radiography with the limb in different positions. This can lead up to a dynamic picture of the actual breaking of the bone and tearing of the soft tissues as it occurred at the time of injury.

TREATMENT OF CLOSED FRACTURES

GENERAL ASPECTS

In planning treatment consideration must be given to the type of patient injured and not merely to the nature of the injury. Age, sex, occupation, general physical condition, temperament, sports, pastimes and social background may all need to be taken into account.

Different fractures occur at different ages, whilst rates of healing and capacity for adaptation vary from young to old. In the child with sufficient growing time before epiphyses fuse, moderate deformity and shortening can be corrected by subsequent growth and remodelling. Prolonged treatment in bed, while irksome to the young, can be harmful to the elderly in whom the decision may have to be not so much their fitness for operation, but fitness to remain in bed with its attendant dangers of bedsores, thrombosis, embolism, incontinence, constipation, urinary retention, loss of circulatory adaptability and pneumonia.

Certain fractures have sex differences: the severe fracture of the pelvis in a woman may be a future cause of difficulty during parturition, whereas in the male rupture of the urethra is a hazard which is almost negligible in the female.

The type of work undertaken will have influence on future employment. In certain jobs early return to work is a probability while in others disability may necessitate change of employment. Self-employed persons may be depressed by the inability to earn while employers may feel compelled to make every effort to resume work even while still unfit.

Patients with different temperaments react in different ways to their injuries: treatment, prospects of a long stay in hospital and rehabilitation, including retraining for a change of employment. The probability of permanent disability may be badly accepted by neurotic and depressive types.

Athletes and professional sportsmen need limbs without shortening, deformity or loss of joint mobility; muscles need to be strong. Musicians need sensitive fingers and free movement of the upper extremities. Hobbies often demand the ability to carry out fine prehensile movements.

The type of dwelling and number in the family may be important factors determining early return home or alternatively the need for a change: for instance to a ground floor flat or a bungalow with no stairs to climb. Elderly people living alone may not be fit to return home for many months. Family responsibilities and worries, or social commitments may weigh heavily in the anxious, over-conscientious or fastidious patient.

TREATMENT OF CLOSED FRACTURES

Wounds heal most easily when divided tissues are accurately apposed and so held in position that disruptive movement cannot occur. Impatience or over-enthusiasm permitting too much stress or mobility too early may delay healing and cause excessive fibrosis or callus formation.

Danis (1949) expressed the opinion that the ideal rigid support for a limb is the intact skeleton and that treatment should aim at its restoration as completely as possible.

The basic treatment of fractures may be stated as (1) reduction to a stable

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position (2) maintenance of reduction and (3) restoration of function (rehabilitation). There are many exceptions: not all fractures need reduction and in some this may be impossible, impracticable, unnecessary, undesirable, or contra-indicated. Otherwise deformity or displacement will need correction; alternatively measures are required to prevent deformity developing later.

Even when the fracture *per se* does not require fixation, the patient has a right to comfort: relief of pain is often the patient's first need. Splintage or support may be desirable or essential in the early stages for this reason alone. Some fractures require support to maintain reduction; others are best held by some form of splintage or fixation to prevent movement of a harmful range at the fracture site; whilst others need no immobilization at all.

Reduction consists initially in making the displaced piece of bone retrace its course to the original position with a minimum of added trauma. The way this is done may vary from the gentle straightening of a limb to reduction under vision after full surgical exposure. The position achieved should be stable and with good bony apposition.

Principles of closed manipulation

Closed manipulation involves making the displaced piece of bone or part of the limb return to its original position by overcoming those factors which are maintaining the displacement. Muscle action is counteracted by anaesthesia and relaxants and gravity by support or positioning. Then the replacement is effected by pulling to correct shortening or impaction, twisting to overcome rotation, pushing to rectify displacement in the long axis, angulation to remove angular deformity and at times to achieve an end-to-end position. The various components of the manoeuvre have to be used in sequence or combined on the basis of the mental picture of the displacement gained from examination of radiographs. Manipulation may reveal the presence of a soft tissue hinge. The procedure is controlled by sense of touch, and the reduction or position of the bones at any time can be confirmed radiographically. Closed reduction needs to be achieved firmly but gently and without jerking. Preferably the reduction should be stable before application of plaster or splints.

Plaster does not immobilize a fracture but it can reduce the mobility to within a safe range compatible with healing. The nearer the bone that the plaster can be placed, the better it holds the position. Padded plasters allow more movement at the fracture. As swelling diminishes the plaster becomes loose and reapplication may be necessary with the attendant dangers of loss of position. Being rigid, plaster can be a source of constriction to a swelling limb. Bandages impregnated with plastics have been used instead of plaster. While these can withstand wetting, they are not porous and the skin may become moist and excoriated.

Following closed manipulative reduction, fractures of the femur are usually better held on a Thomas splint with some form of traction to hold length and position.

Until recent years the closed treatment of fractures was standard practice, deformity being corrected by manipulation followed by splintage or plaster, whilst operation was mainly reserved for compound fractures and certain special indications. The introduction of the "no-touch technique" aided by modern anaesthesia and blood transfusion has opened a new era of fracture treatment and made prac

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tractable the internal fixation of many of the more severe bony injuries. Nevertheless progress has been slow and for a time the only common open interference was the use of skeletal traction by taut wires or rigid pins. More recently still and especially since the introduction of antibiotics and new metals the trend has been continued towards the open reduction and fixation of the majority of difficult fractures. Nowadays the emphasis is changing to rigid fixation as a means of restoring skeletal anatomy and avoiding many complications or undesirable sequelae.

Most closed fractures including those of ribs, clavicles, the pelvis, vertebral bodies and almost all fractures in children are normally treated without operation. Others, such as Colles' fractures and those of the carpal scaphoid are rarely considered to need surgery whilst certain other fractures, notably those of the calcaneum and certain types of Pott's fracture are often considered to be better treated conservatively.

In spite of a trend towards open reduction and rigid internal fixation those fractures which will unite in reasonable time without significant disability when treated by closed methods, should still be so treated. Operating theatres and techniques are never perfect and all open reductions carry a small but definite risk of wound infection.

If experience has shown that a particular type of fracture is notoriously unstable or likely to be followed by joint stiffness and disability when treated in plaster then operation, especially rigid fixation should be considered. In certain fractures—for example those of the forearm in adults and many fracture-dislocations of the ankle—where it is known that open operation can give better results non-operative treatment should be discarded.

Most fractures of the femoral neck have been subjected to operation since the introduction of the trifin nail and more recently nailing of pertrochanteric fractures has largely replaced their treatment by splintage and traction. On the other hand a few fractures, especially those of the radial head, may have been too generally regarded as needing open interference. In all fractures the probable duration of alternative treatment and the resulting degree of disability should be weighed against the risk of operation which should never be undertaken when its prospects of success are poor.

Operation

When operation is needed it should be undertaken at the optimum time. Early intervention should be carried out within a few hours before "fracture blisters" form, before bloodstained tissues become friable from oedema and skin healing less certain. Patients with injuries should not have to wait until a list of other emergencies has been completed.

If practicable, exposure of the fracture should be through the "hidden wound" rather than through a classical exposure somewhere else. Often the exposure which would be correct for a "cold" orthopaedic operation will cause additional damage to bone or muscle, in an injured patient.

Fixation

Occasionally open reduction is all that is needed. Sometimes the presence of a soft tissue hinge with a transverse fracture will allow a stable position to be held

by a plaster cast sometimes the end of one fragment can be jammed into the other. Open reduction is however usually followed by internal fixation.

Circlage wiring combined with external splintage or a plaster cast is suitable for some oblique or spiral fractures especially when the complexity of the fractures makes the introduction of screws impossible. Such wiring can be modified to encircle the bone more than once or pass through drill holes before it is directed around the bone. Manoeuvring the wire into place by special forceps or a modified aneurysm needle does not require extensive soft tissue stripping. The hour-glass constriction leading to weakness and fracture at the site of circlage wiring results from chafing when movement follows inadequate fixation. It can also occur in the absence of such movement when the wire tightly constricts the periosteum from without. Encircling metal is clearly unsuitable for transverse fractures.

While two or more screws can give a firm stable fixation after reduction a single transfixion screw is rarely adequate to hold the oblique or spiral fracture. Movement can occur by cutting out of the screw or bony rotation around the screw.

Plating is the traditional and until recently perhaps the most used method of internal fixation. The plate should be as strong as possible and of adequate length. The screws should, if practicable, avoid fracture lines for fear of causing displacement and should penetrate both cortices to give the firmest fixation of plate to bone. Before main fragments are fixed together by the plate other sizeable pieces of bone, especially butterfly fragments should be screwed to either of or both the main fragments.

Intramedullary nails have a valuable place in treatment, particularly for the femur but rotation can often occur about a longitudinal axis. Like the Smith-Petersen nail, the Küntscher nail becomes loosened and after 3 weeks can be removed without the aid of a mechanical extractor. The rigidity of fixation is increased by an accurate fit or cortical contact.

Many other methods of bone fixation have been used including nuts and bolts, lag screws, staples and spikes. On rare occasions primary bone grafting is advisable. Bony fragments can be controlled by Kirschner wires or Steinmann pins incorporated in plaster or held by retentive apparatus consisting of metal bars and clamps. Pins may be single or double or of a special pattern such as those designed by Anderson. In general these methods have a limited application but can be of particular value in treating the extraordinary injury. In some circumstances methods of fixation may require to be modified or combined.

Treatment should be tailor made to suit the individual injury. Surgery aims to close the whole wound, including the osseous tissue and to maintain apposition of the realigned fragments strongly enough to provide splintage when necessary. In this sense bony fixation is a form of suture and healing takes place across the smallest possible gap. Stable reduction by internal fixation of the fracture allows torn soft tissues to lie in apposition. Alternatively soft tissue repair can sometimes result in stability of a fracture so that internal fixation becomes unnecessary.

Technique

Open reduction converts a closed bony injury into an open fracture and infection must be avoided. Operation under an umbrella of antibiotics carries the hazard of tolerating relaxation of well-established aseptic methods.

While the classical orthopaedic "no touch" technique is probably still worth

TREATMENT OF CLOSED FRACTURES

while it should be adapted to particular conditions. The finger is a sensitive and delicate tool at times the safest. Introducing a finger covered by a freshly sterilized or specially cleansed glove allows a surgeon to gain information that would otherwise be obtainable only by wide dissection. Taking a limb apart for information is not justifiable.

In instrumental surgery the operation field is not concealed by hands and the theatre team can observe the steps of the operation and be able to anticipate the surgeon's requirements. In the hands of a practised team the operation is neater and slicker.

Gentleness is all-important. Whatever the technique tissues must not be traumatized. Rough handling can lead to sloughing of skin and may cause death of deeper tissues with delayed healing and excessive fibrosis in addition to predisposing to infection. There is no objection to the use of scissors provided they are sharp. If an operation demands more than one exposure the risk of infection can be reduced if each wound is regarded as a fresh operation with changes of gowns, gloves, towels and instruments. This elaboration may not always be practicable but is worth aiming at.

The metals in plates and screws should be discarded.

The dictum attributed to the late Sir David P. D. Wilkie is apt. In operating suppress every manoeuvre which is not definitely of value.

Post-operative care

A plaster may not be required. Danis (1949) used short semi rigid splints instead of plaster so as not to add "the illness of immobilization to the injury of accident". Other surgeons use neither plaster nor splints after rigid internal fixation.

It is our experience that after rigid fixation plaster puts the injured and incised soft tissues at rest and allows rapid healing with a minimum of fibrosis. After the tibia has been plated, many patients lie with the limb externally rotated, the knee flexed and the foot in equinus if a cast is not used. Apprehension and discomfort may last for days. On the other hand some patients are comfortable and confident from the time of operation. Active movements of muscles and joints are soon restored and stiffness does not develop. Sometimes swelling causes tension in the suture line leading to pain. Cutting in of sutures and localized sloughing, all of which will be hidden under the plaster. The even pressure of an unpadded cast can prevent swelling and its undesirable complications. If the toes are not swollen it is unlikely that there is any swelling of the limb. Many patients treated by rigid plating and plaster are comfortable from the time of operation. They can get up on crutches early and be discharged home after a few days. If the fixation is adequate the plaster can be discarded after 3 weeks. Exercise soon restores muscle power and joint mobility.

Delayed operation

When operation has been delayed for one reason or another the skin may need special cleaning. Tissues will be found oedematous, somewhat rigid but friable with evidence of incomplete healing in some areas. A successful reduction and plating may now demand uniform shortening of the bone ends to achieve a fit, or conversion of the fracture to a step-cut before plating.

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Wide stripping of bone and removal of callus is not good surgery. Forcing or levering the bone ends into apposition results in difficulty in closing the wound from loss of elasticity in the soft tissues. Suture of the skin under tension may lead to sloughing of the skin. Infection is then likely and the bone may be exposed for a long time before healing takes place by granulation and fibrosis. If the bone becomes infected final healing may take years and a severely disabled limb will result unless circumvented by amputation.

When the radiograph before operation shows patchy decalcification the bone may be soft and difficult to handle. Plating may not be possible. At operation the patchy decalcification is seen as a magenta mottling and appears to be granulation tissue replacing bone.

When delayed plating is carried out without excessive stripping of bone and closure of the wound without tension it can result in quite rapid healing of the fracture. For example, sound clinical and radiological union of the tibia in 16 weeks.

OPEN FRACTURES

Open fractures are urgent surgical problems demanding treatment without delay. Whilst operation converts closed fractures into open injuries, the aim of surgical interference is to convert open fractures into closed injuries.

The name "open" or "compound" fracture emphasizes the bony damage, yet it may well be that the associated soft tissue injury will be the factor finally deciding the fate of the limb.

Today the increase in mechanization, particularly the advent of the motorcycle, has led to an increase in the number and severity of open fractures, especially of the tibia and fibula in males between 16 and 40 years of age. Many of the riders of these machines hide their heads in safety helmets and with a false sense of security forget their limbs have been left exposed.

The risk of infection from such open wounds is well recognized. Equally important is the occurrence of damage to the blood supply distal to the fracture site. Attention must also be paid to injury to joints, nerves, muscles and tendons.

Types of fracture

Fractures compound from within result in a puncture wound, a small wound with projecting bone or in tearing of skin and soft tissues over a sharp bony prominence due to angulation or torsion of the limb. When the fracture is compound from without the wound may be small and incised but usually it is ragged. The skin damage varies considerably with respect to undermining, flaying (degloving) and skin loss. As a general rule, the greater the skin damage the more the associated injury to other soft tissues and the more severe the fracture. In a crushed limb all tissues may be severely damaged.

Some fractures are compound from without and from within, the bone end being protruded by a longitudinal compression force and afterwards retracted. In these, soft tissue damage and soiling are usually greater than the size of the skin wound would suggest.

Certain fractures appear to be closed but should be regarded as potentially open. Death of skin is produced by pressure against the underlying bone or by tension over a bony ridge or prominence, the cutis vera having split, the intact epidermis may be the only barrier between the fracture and the outside.

OPEN FRACTURES

The severity of open fractures varies from a minor fissure fracture exposed by a clean incised wound to the severely crushed limb needing amputation on sight. The intermediate grades of injury require to be treated according to their needs. The treatment can only be decided on the basis of a full clinical diagnosis modified by findings at operation.

Clinical examination

Examination of the limb should note the state of the circulation, the extent of wounding and the bony damage. If a limb is to be saved there must be an adequate circulation beyond the level of injury. There may be no obvious circulation in the injured extremity of a shocked patient, but adequate transfusion may restore sufficient circulation to warrant an attempt to save the limb.

The extent of wounding should be fully noted and the probability of wound closure by suture or grafting assessed. Skin is the best barrier against infection and a split-skin graft, or even replaced flayed skin may serve for a time.

Injuries to joints, nerves, muscles and tendons need to be assessed in relation to restoration of adequate function. Bony damage is estimated by inspection and gentle manipulation supplemented by radiographs, preferably taken after application of temporary plaster splints, which allow movement of the limb without pain or detriment. The prospects of stable reduction with or without internal fixation should also be estimated, remembering that the bony injury is always greater than shown radiographically, especially as regards longitudinal splitting of main fragments.

If amputation is considered, it should be remembered that prostheses for lower limbs are more satisfactory than artificial arms. Preservation of a movable joint is always worth while and even a small part of a hand may be useful, because of its function as a sense organ.

Lines of treatment

The possible lines of treatment include (1) repair of the injury and preservation of the limb (2) surgical exploration of the limb before deciding whether to attempt preservation or not (3) attempted preservation with possibility of amputation later and (4) amputation.

For the preservation of a limb, skin cover and circulation must both be adequate and the fracture must be capable of stable reduction which can be held by splintage internal fixation or a combination of the two. Preferably the skin should be sensitive and there should be a good prospect of useful function.

When indicated, immediate amputation gives a better stump than delayed amputation because sloughing is less likely and the danger of infection reduced. The stay in hospital is short and return to work can be early. Even a patient over 60 years of age can learn to use an artificial leg, especially after below knee amputation.

A futile or unsuccessful attempt to save a limb often means a long stay in hospital, multiple operations, prolonged sepsis and disuse of a limb. After this, amputation and fitting of a prosthesis are too often followed by a disappointing result. The patient is not easy to rehabilitate, may be disinclined to return to work and may not return happily to the family circle.

Once a decision has been made the plan for dealing with the limb should be explained to the patient (even a child, if old enough to understand) and to the relatives so that it is clearly understood what is entailed in saving the limb especially as regards rehabilitation. Agreement to amputation should be obtained beforehand in case such treatment should be indicated after further examination under anaesthesia. The necessity for amputation should be made clear including the probable level and a provisional date of fitting a prosthesis.

Pre-operative treatment

Dressing of the wound control of bleeding and splintage of the limb are needed. Plaster slabs massaged into place with the limb lying on a mackintosh pillow make admirable splints which can be bandaged in place as soon as setting has occurred. The application should be painless and is better carried out before the patient is moved to the radiological department or elsewhere.

Prevention and treatment of shock are discussed in Chapter 5. If needed a small dose of morphine or pethidine can be given intravenously for relief of pain.

Operation

The operation should be carried out as soon as the patient's general condition permits. A tourniquet should be used only when soft tissue damage is of a minor character. Without it, the vascularity can be better seen in soft tissues, with the exception of flayed skin in which the absence of circulation is no guide to survival. Further injury to major vessels becomes obvious sooner.

The tourniquet is better avoided in aged patients particularly if the arteries are calcified or if there is a history of claudication. If a tourniquet is needed, the pneumatic type is preferable. When applied to the thigh it should be in the upper third where the artery is protected by muscle and not in the middle third where the artery is probably more vulnerable to pressure. Constriction at this level ties down the rectus femoris muscle which may be damaged by passive flexion of the knee. This movement is limited by the tourniquet and positioning of the limb may be difficult.

Bleeding may sometimes be profuse at the site of the operation when a tourniquet is not used, and the transfusion rate will need to be increased. If much bleeding is expected transfusion should be started before the limb is cleansed. After cleansing of the limb wound excision should be carried out as in any other type of open wound.

In view of the importance of skin closure minimal excision should be carried out at the start. Adequate excision of dead, devitalized and dirty muscle is the one practicable defence against gas gangrene. Deep sutures are undesirable but severed nerves or tendons can be "hitched" with black suture material for easier identification at a later date. Suture of muscle and fascia over bone can provide a vascular bed for a split skin graft. Suture or grafting of main vessels may sometimes help to preserve a limb which otherwise must be amputated. After excision, joints need closure by suture of capsule and ligaments.

The fracture

Soiled bone needs to be cleansed. This should be carried out with a minimum of damage to attached soft tissue. Cleaning may be completed by sparingly removing tiny flakes with mallet and chisel or nibbling away with forceps.

OPEN FRACTURES

All fragments large enough to be handled should be preserved even if completely detached. Those with soft tissue attachments need handling with care to preserve any remaining blood supply.

Extension of the wound of injury to permit fixation should have regard to maintaining a good skin circulation. The line should not of necessity follow an accepted exposure of a particular long bone. The exposure should preserve any soft tissue attachment to bone especially a soft tissue hinge which will contribute to stability of reduction. During internal fixation, further separation of soft tissue from bone should be avoided as far as possible. Detached bony fragments should be replaced after suitable cleansing or sterilization. They may even be boiled. Larger pieces may need screwing or wiring in position. Stable reduction should be the aim in treating the open fracture and there is no objection to the introduction of metal. Rigid internal fixation is better than plaster or splints and permits of early mobilization.

Firm internal fixation is necessary if pressure dressings are to be applied over split-skin grafts or when flap grafts are used especially cross leg flaps. Under split skin, replaced flayed skin or flap grafts intramedullary nails may be preferred to plates and screws which are better covered by full thickness visible skin.

Closure

Under war conditions there was much to be said for delayed primary suture for open fractures especially when patients were moved from place to place. In civil practice primary closure of skin is the ideal. Delayed primary suture is reserved for the occasional amputation stump otherwise it is used as a valuable compromise. There is no justification for secondary suture or leaving a wound to granulate. Such wounds always become infected, healing is slow and excessive fibrosis develops before epithelialization from the skin edges brings about skin cover. The thin skin which results is often tied down to bone and the fibrous tissue interferes with muscle movements. Ultimate function is poor and the epithelialized area readily cracks and ulcerates.

The skin should be closed without tension and for this purpose limited undermining, local rearrangement or grafting may be needed. In some instances local rearrangement and suture of skin with split-skin grafting of areas away from the bone as for example relief incisions, may be needed. Sometimes flayed skin should be replaced sutured, and supplemented by split-skin grafting as required. In a few instances conversion of a fracture to a step cut will shorten the limb a little and allow closure of a partial circumferential loss. (This manoeuvre may also allow of nerve or arterial suture.)

Skin loss may be replaced by a full thickness flap graft, for example a cross-leg flap after firm preferably rigid internal fixation. This may be needed when there is bone exposed with no prospect of closure by other means. It should not be undertaken lightly for it adds considerably to operating time. Once the flap is in position skilled dressing and plastering must follow. Such a prolonged operative procedure would be out of the question in a patient in poor general condition from multiple injuries. It may be contra indicated on account of age or infirmity or where arthritis would make the fixation intolerable. Whatever the method of skin closure employed, dressing and plaster or splintage will be required in most instances for at least 3 weeks. Split-skin grafts need

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properly applied pressure dressings. At the end of the operation full details of the damage sustained should be recorded, together with a full account of the repair effected and proposals for future surgical procedures likely to be needed for maximum restoration of function.

Post-operative treatment

In favourable cases post-operative progress should be as uneventful as in closed fractures subjected to open repair. The injured limb should be elevated, kept cool and the circulation observed.

Split skin grafts need early inspection if haematoma formation is anticipated, and a flap graft needs to be looked at even earlier to deal with any kinking that may have occurred at the base. Flayed skin does not all die: the total loss may be less than half. Excision and split-skin grafting will be needed once the dead areas are clearly demarcated. Separation of a flap graft should rarely be undertaken before 3 weeks. The interval may be much longer when the original attachment has been somewhat limited or when the graft has been sutured primarily to skin with an impaired circulation. Plaster can be removed after 3 weeks, sutures taken out and remedial exercises started to restore mobility to muscles and joints provided that the skin has been sutured without tension and rigid internal fixation has been achieved.

Removal of plaster and sutures in other cases will depend on the merits of the particular injury. Full activity must await sound healing of skin and bone and a successful outcome of any subsequent operation found to be necessary.

Post-operative complications

Haematoma formation may occur following any operative treatment of a fracture, but is more likely after an open injury especially with severe bony damage. A large haematoma with only moderate oedema of the skin can result in cutting in of sutures and localized sloughing. Progressive swelling of digits should suggest such an occurrence and the wound will need inspection by removal of plaster or by cutting a window if need be. Post-operatively some sloughing may be encountered even in closed fractures but it is more likely in more severely injured limbs. After open fractures sloughing of damaged skin is not uncommon: the area may be quite small or may be large enough to demand flap grafting as a secondary procedure.

Exposure of a plate may result from sloughing of skin. If it is limited in extent suitable dressings can prevent infection and the exposed plate will be covered over by granulation tissue and the spread of epithelium. Otherwise the appropriate plastic procedure will be indicated.

Local infection must be watched for: if suspected, the wound should be exposed and a swab taken for culture. While awaiting bacterial culture and sensitivity tests a wide-spectrum antibiotic should be given. Afterwards the appropriate antibiotic should be given in adequate dosage. The infected haematoma or abscess will need to be opened. Deep infection around the fracture is to be feared and must be treated vigorously by surgery and antibiotics if osteomyelitis is to be prevented.

COMPLICATIONS AND SEQUELAE OF FRACTURES

Joint stiffness

Normal joints do not become stiff in plaster but the age at which joints cease to be normal is variable. Degenerative changes have been seen in the shoulder and

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sacro-iliac joints in patients as young as 30 years of age. At the same age symptomless erosion of the articular cartilage of the patella has been described. Limited joint mobility may result from any of the following circumstances: (1) direct injury causing intra articular adhesions, fibrosis of ligaments and capsula and even bony or fibrous ankylosis. (2) injury jarring or traction on a limb resulting in clinical deterioration in a pre-existing osteoarthritic or otherwise abnormal joint. (3) adhesion of muscle at the site of injury or muscle shortening. (4) impaired muscle power due to direct injury nerve damage or disuse atrophy. (5) fibrosis of muscle and irreversible changes due to ischaemia from arterial damage. (6) involvement of a joint in the zone of inflammation or reaction associated with a fracture or other major injury.

Oedema following Injury

Apart from medical causes, such as cardiac failure which may have developed or progressed during treatment, swelling in dependent limbs must be attributed to the injury. It is seen most often in the lower extremity following the removal of plaster after a severe ligamentous or bony injury below the knee. Thrombosis of the deep veins of the lower limbs may have developed during treatment. The condition is characterized by pain in the calf and swelling of the toes. Pre-existing varicose veins seem to be aggravated by the injury and use of a plaster cast, probably through thrombosis of the deep veins.

Other causes of the swelling are obscure but may include extensive damage to lymphatics with diminished drainage, impaired function and lack of muscle contractions resulting in a sluggish venous return and possibly the persistence of increased capillary permeability which was a feature of the early inflammatory reaction.

Oedema of the leg after removal of plaster is common in patients over 30 years. As a rule the more serious the injury and the longer the immobilization the greater and more persistent the oedema. If the swelling is the result of otherwise symptomless deep-vein thrombosis anticoagulant therapy should be used as a prophylaxis. The value of such treatment needs to be investigated but medication should be started early if it is to be effective. Elastoplast zinc paste bandages and crepe bandages can help to prevent gross and painful swelling. At first when the patient is not walking the leg should be elevated. Later periods of rest in elevation will suffice. Remedial exercises and contrast baths are helpful. When the oedema persists a well-fitting elastic stocking may be of value.

Decalcification

Decalcification following fractures is primarily a radiological sign. It occurs mainly in adults and is seen more frequently distal to the fracture and in the lower rather than the upper extremity. It consists of a patchy loss of density involving the lower forearm bones, the carpus and metacarpal bases in the upper limb, the lower ends of the tibia, tarsal bones, metatarsals and phalanges in the lower limb. The extent to which it is seen depends on the frequency with which radiographs are taken. It can involve the affected bone above the fracture and is often seen in bones higher up the limb so that its recorded predominance in the distal portion may be due to the fact that radiographs are more frequently taken of the injured parts. The clinical and prognostic significance of this type of decalcification has not

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been established. Pathologically it consists of a loss of bone substance not merely of lime salts. Some observers consider that it indicates liability to swelling and joint stiffness although these may be due to coincidental factors. It is not necessarily associated with pain. Often it clears up uneventfully but may appear to progress after removal of plaster and union of the fracture.

Other forms of decalcification are attributed to disuse, paralysis, senility and inflammation and tend to be uniform. The cause of this patchy type is not known but disuse and inflammatory reaction to injury may be among the causative factors. There may be vasomotor influences resulting from minor damage to nerves or vessels.

The condition clears up slowly. Rest and physiotherapy may help but plaster may be inadvisable. Weight bearing is contra indicated when the condition is marked in the lower limb.

This type of bone atrophy probably does not progress to that described by Sudek with its dramatic clinical and radiographic changes. When healed the bone shows a coarser trabeculation than that in the uninjured limb.

Delayed union

Today many fractures are caused by greater violence than in the past. The result is greater injury to the tissues and considerable damage to blood vessels with a diminished blood supply to bone. Further stripping of periosteum around the fracture reduces the cortical blood supply especially when the injury has cut off the supply from the nutrient artery. The extent of death of bone at the fracture may be considerable. Callus may not form and delayed union may well result. The delay may be so prolonged as to constitute non union without callus. Open fractures show a greater incidence of delayed union especially when complicated by infection or breaking down of the wound.

Movement at the fracture site beyond a safe range may result from poor immobilization due to inadequate splintage, soft or loose plaster and ineffective internal fixation not supplemented by external support. The result is the breaking down of forming callus through repeated small traumas, each incident being followed by attempts at repair. In an effort to bridge the gap more and more peripheral callus forms and may bring about immobilization if formed in sufficient quantity and strength over a sufficiently large area. On the other hand, if the attempt proves abortive non-union with much callus will result.

Union can be delayed by removal of plaster or splints for further manipulation after the end of the second week even the minor adjustment of alignment by wedging the plaster after that time can have a similar undesirable effect. Too early removal of external fixation can result in the breaking down of a healing fracture with delay in uniting.

True distraction as distinct from the radiological appearance of a gap between the bone ends in an oblique view can only happen when there is extensive soft tissue damage. It indicates the severity of the injury and should suggest that healing will be slow especially when the gap must be filled by callus.

Infection after operation causes delayed union only when it involves the fracture site. Superficial infection may cause little or no delay. Corrosion of metal may interfere with the progress of union.

Delayed union is primarily treated by removing the cause. Movement at the

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fracture site must be stopped and infection eradicated Corroding metal must be removed and sometimes needs to be replaced by plates and screws of a safe type Inadequate blood supply is not easily treated time is needed The fracture area must be protected against harmful movement until revascularization of the necrotic cortical ends has occurred Only then can there be reconstitution by new bone which bridges the gap between the fractured cortices

Replacement of a thin adherent scar overlying a fracture by a full thickness pedicle graft can stimulate healing in the underlying bone presumably by bringing a fresh blood supply to the fracture site

When delayed union is ignored and adverse influences are allowed to continue non union is certain although removal of causative factors even at a late date can be followed by union in a fracture with many of the features of so-called established" non union

Non-union

Non union may be presumed when it is clear that in spite of maintenance of reduction, clinical union is unusually delayed and there is lack of radiological progress over a reasonable period This may result from failure to diagnose and treat a delayed union.

Non-union may occur without callus, or with excessive callus, or there may be irregular formation not bridging or ensheathing the fracture There may be fibrous or fibrocartilaginous material between the bone ends later the formation of a bursa due to repeated movement may produce a pseudarthrosis

Operation is usually indicated and bone grafting is the treatment most often employed. Grafting as practised by Phemister (1947) is favoured by some surgeons others prefer sliding or onlay grafts The latter can be massive giving a degree of rigidity but are rarely used Smaller grafts can be supplemented by plating or nailing. The Phemister type of graft gives no fixation at all but the success that follows its use suggests that its presence stimulates union Like other bone grafts it provides a scaffolding on which new bone can spread from one fragment to another Almost all grafts die and bone that has been boiled forms a suitable graft Even decalcified bone has proved satisfactory when used experimentally The dead bone of a graft will fuse to living bone at either end remaining white and avascular elsewhere for a long time if it is in contact with fibrous soft tissue with an impoverished blood supply It can also be claimed that a graft can maintain, in the fibrous tissue between the bone ends a gap across which bone can grow when it was presumably unable to pass the barrier of fibrous tissue

Fibrous union of the radius has been successfully treated by rigid plating (see Chapter 10) The author has also successfully treated fibrous union of the medial malleolus by driving two pegs of bone cut from the adjoining tibia through the disc of fibrosis from malleolus to tibia. Jeffery (1958) has reported similar gratifying results from bone pegging for ununited fractures of the lateral condyle of the humerus

Non union of the tibia with pseudarthrosis and the typical "elephant's foot" formation of bone due to heaping up of excess callus, is usually associated with a united or intact fibula. Osteotomy or resection of a portion of the fibula will permit apposition of the ends of the tibia. After paring, rigid plating is to be preferred to bone grafting and union will result.

PRINCIPLES OF THE TREATMENT OF FRACTURES

For non union with little or no callus bone grafting will need to be preceded by freshening of bone ends or removal of scar tissue. Occasionally hard & obviously avascular bone is present and needs excision. The resulting gap has to be bridged by a cortical graft and the remainder of the gap filled by cancellous bone. Plating will add rigidity to the repair. Although union is rapid consolidation is slow and the union will be brittle for about 2 years. During that time protection is essential. Intramedullary nailing is sometimes a better alternative.

Following grafting of any kind splintage or plaster is necessary until union has occurred but it is not needed if rigid plating has been effected at the time of operation.

Post-traumatic arthritis

In some patients pain and increasing stiffness develop in a joint near a fracture. The interval may vary from a few months to several years. The condition is progressive and in time the radiographs will show typical osteoarthritic changes. This is in contradistinction to the residual joint stiffness due to loss of elasticity of the soft tissues around the joint of an injured limb. In this condition rehabilitation brings about improved joint function until progress ceases and the range of movement remains unchanged.

The causes of post-traumatic arthritis are as follows:

(1) Direct injury to a joint with disruption of the articular surface resulting in bony steps. After an interval typical osteoarthritis develops. The condition is illustrated in the fracture of the lateral femoral condyle allowed to unite in deformity and with a moderate genu valgum.

(2) Static causes resulting from union of a fracture with angular or rotational deformity leading to changes in the weight-bearing line of the limb and uneven stresses on the articular surfaces of neighbouring joints. Irregular wear of articular cartilage occurs leading to osteoarthritis.

(3) Aggravation or exacerbation of a pre-existing osteoarthritis or degenerative arthritis. For example, the elderly patient with a fracture of the femoral shaft treated by traction and splintage will complain of pain on removal of traction and active joint movements. The knee shows swelling, effusion, crepitus on movement and tenderness especially over the medial ligament. Symptomless crepitus may be present in the knee.

(4) Cartilage atrophy as a cause is controversial. The bone of a broken limb undergoes patchy decalcification and disuse changes can occur in all tissues if immobilization is prolonged. Muscle wasting can be seen, and laxity of ligaments can be demonstrated. Atrophy of articular cartilage with thinning out and possibly fibrillation can be presumed. Resumption of active movements and especially weight bearing could lead to ulceration of the cartilage. If this interpretation of events is correct mobilization of the limb should be cut short by using rigid internal fixation which would permit early active movements.

REHABILITATION

Rehabilitation usually means physiotherapy, remedial exercises, occupational therapy and industrial retraining, aimed at restoring the injured limb to function and the patient to health and vigour. Although patients should be encouraged to contract muscles and move joints this must not be done "at all costs". In the case

REHABILITATION

stages of treatment exercises must not cause pain and patients should be told that any activity causing pain may be doing harm. Massage may be of no value in some injuries and may be definitely harmful in others particularly those in the elbow region.

Following injuries to the knee, muscle inhibition may be observed in the hamstrings during remedial exercises. When active movement reaches a certain point, contraction of the hamstrings ceases. Passive movement can continue through a greater range but may still cause pain. In the author's opinion, the loss of active contraction can be regarded as a defence against movement through a dangerous range and may be of a reflex nature. A similar inhibition may be seen in the triceps muscle in elbow injuries. Injuries to the shoulder may be followed by an inhibition which results in physiological subluxation of the humeral head being shown by radiography to be low in relation to the glenoid fossa. Muscle inhibition is an indication of incomplete healing and necessitates a reduction of activity or even rest. As healing proceeds muscle action is restored and the condition disappears.

REFERENCES

- Danis, R. (1949). *Theorie et Pratique de l'Osteosynthese*. Paris: Masson et Cie.
Jeffery C. C. (1958). *J Bone Jt Surg.*, 40B, 396.
Phemister D. B. (1947). *J Bone Jt Surg.* 29A, 946.

CHAPTER 10

FRACTURES OF LONG BONES

F G BADGER

INTRODUCTION

THE feature of long bones is their length and the surrounding soft tissues correspond in length to the skeletal parts they invest. Whilst these conditions obtain limb function is likely to be normal provided that all tissues including bone, are healthy.

The architecture of a long bone varies over its length. The ends are composed of a thin layer of compact bone covered with articular cartilage and deep to this cortex is spongy bone with an elaborate trabecular structure. The shaft consists mainly of compact cortical bone with a little cancellous bone in the centre. Between the articular area and the shaft the cortical bone gradually thickens and the spongiosum diminishes. In weight bearing bones the cortex tends to be thicker and in the femur and tibia it is denser and thicker than in any other bone in the body. Healing is influenced by the structure and vascularity of bone: the compactness and relative avascularity of cortical bone leads to slow end-to-end healing, whilst the open structure and better blood supply of cancellous bone favours union in shorter time.

In the neighbourhood of joints, cancellous bone acquires a blood supply from the arterial circle. Nutrient arteries enter the shafts of long bones but the periosteum which receives most of its vessels from neighbouring muscles is the main source of the blood to at least the outer half of the cortex. Healing is slower when the soft tissue mass around the bone is significantly damaged and particularly when the periosteum is stripped. On the other hand, intramedullary nailing must frequently disrupt the nutrient artery but apparently does not interfere with union.

Compression

Charnley (1953) and Danis (1949) described healing of apposed bone surfaces as occurring more rapidly and completely under compression although Charnley found this true only for spongy bone. He claimed that compression is the important factor and that it facilitates the rapid fusion which follows his method of arthrodesing joints. He did not believe that his apparatus primarily brings about firm fixation although the incidental immobilization produced is excellent. Others believe that "complete immobilization is essential for rapid complete bony union or fusion. Danis (1949) insisted that compression stimulates healing of all fractures but he combined it with rigid fixation in such a way that the effects cannot be distinguished. It can be argued that normal bone is subject to compression and that this environment is necessary for normal metabolism and reproduction of bone.

THE LOWER LIMB

cells. Certainly the muscles by their tone bring about a degree of longitudinal compression while the periosteum could compress the bone transversely. Long bones usually unite without compression or rigid fixation although some times shortening, displacement and excess callus occur. Oblique and spiral fractures of the lower tibia often unite rapidly in plaster with a little shortening especially in circumstances which allow bony contact of fracture surfaces over a large area.

Union in children

In children when a long bone unites and produces shortening rapid growth takes place and loss of length is corrected sometimes overcorrected. How this happens is not known but it is possible that the bone grows until the tension in the soft tissues especially the muscles has been restored to normal. Hyperaemia can cause rapid growth of a bone in a child in the event of slow union or multiple manipulations. Hyperaemia might stimulate excessive growth. Angulation up to 30 degrees can often be rectified by normal growth and remodelling but the process may be slow and extend over 2 years or more. If there is not sufficient time before union of the particular epiphysis, deformity will result and require correction.

Puberty and maturation of male and female children are believed to occur earlier than was once thought normal (Report 1958). Nowadays radiographs of 15-year-old girls usually show fused epiphyses, some of them may be fused at 13 years. In many males epiphyses are fused at 17 years of age.

Delayed union and non union are rare in the child in whom bone growth and periosteal osteogenesis are active.

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In the lower limb length and stability are of primary importance. Range of movement is less essential because in all joints it exceeds that necessary for ordinary usage. It is usually accepted that half an inch of shortening in a lower limb is of no consequence but this is by no means always so. Some patients complain bitterly of less than half an inch of shortening while other patients get about happily with one and a half inches or more. Material shortening is usually compensated for by tilting the pelvis and this may persist for many years without symptoms or complaint. Later low back pain may develop due, it is claimed, to osteoarthritic changes in the lumbosacral joint. Be that as it may many patients who have had no relief from physiotherapy manipulation and spinal supports have been rapidly rendered symptom free when the discrepancy in length has been made good by raising the appropriate shoe. Occasionally shortening is corrected by walking on the toes.

Union with angulation can result in stresses on joints being exerted through abnormal axes with uneven weight bearing and irregular wear leading eventually to osteoarthritis. The changes may be limited to part of a joint—for example the inner or outer compartment of the knee—and resemble those developing in genu varum or valgum. Charnley (1953) claimed that these changes are of an adaptive nature—a condensation of bone in response to stresses—an example of Wolff's law. Nevertheless typical osteoarthritis develops and causes symptoms.

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Rotational deformity after fracture, unless of minor degree cannot be compensated for in walking by rotation at the hip. Accordingly the greater part of the weight is thrown on the outer or inner border of the foot with consequent discomfort particularly if the foot itself is stiff. Abduction or adduction deformity below the knee or in the ankle can result in weight being unevenly distributed at the ankle or foot. Angulation of the lower femur producing backward bowing can result in a genu recurvatum deformity with undue strain on the posterior ligaments of the knee joint giving rise to pain and disability.

Fractures of the tibia and fibula

It was once generally accepted that a broken leg required splintage for 8 weeks but the average time to union in a large number of cases has increased until it now exceeds 20 weeks in the adult. Mechanical reduction, advocated by Böhler (1938) was in vogue at the time when the increased time to union was noticed.

Distraction was blamed but many of the fractures were not subjected to skeletal traction and more accurate observation has shown that the increase was not associated with the method of treatment. The explanation lies probably in greater violence causing worse fractures much of it results from the increased speed and number of road vehicles especially motorcycles, and the increased mechanization in industry.

Fractures of the upper third of the tibia, where there is much cancellous bone, are often said to unite quickly and non-union is said to be infrequent, but this is more apparent than true because these fractures are relatively uncommon and in fact non union occurs as often as in shaft fractures lower down.

Some tibial fractures are still sustained as they were in the past and still heal quickly when treated in the old fashioned way. Others may require different treatment nowadays more tibial fractures are being treated by open reduction and internal fixation. By this means deformity and shortening are prevented. A plate, for example, should hold the fragments in apposition and must be long enough and strong enough to prevent movement from breaking down the zone of healing. When movement is repeated it leads to an abundance of peripheral callus although not necessarily to delayed union.

Before unprotected weight bearing is permitted, radiographs should show either that the fracture lines are obliterated or filled in by bone or that the whole fracture is surrounded by dense callus. Where only part of the fracture line has disappeared, union cannot be considered sound unless the unhealed part is bridged by a sufficiently strong buttress of callus. The fracture area should not be significantly swollen or tender. Pain should be absent when angulation is attempted by the surgeon and weight bearing should not result in pain, warmth or tenderness over the fracture site. Not all patients have the same appreciation of pain and some disregard the aching and continue to bear weight. The fracture may then break down, mobility increases and a pseudo-arthritis develops, apparently painless except after unwonted exertion or in inclement weather. This condition of "functional non-union" is in contrast to non union which prevents the limb from being used and for which treatment is needed to correct the failure of bony healing. Occasionally functional non union in the neighbourhood of a joint rendered stiff by the injury may be an asset.

If the aim of sound union without shortening or deformity cannot be fulfilled loss of length can be corrected by raising the boot or shoe. However rotational and angular deformities lead to awkward gaits and joint changes. Radiological deformity may be apparent while the functional line is normal. The upper and lower articular surfaces may be at right angles to the weight bearing axis of the tibia although the intervening bony fragments may be irregularly disposed.

Cross-union between the tibia and fibula rarely causes symptoms but lack of mobility of the tibiofibular joint may result in loss of part of the range of ankle movement usually with impaired dorsiflexion. If the fibula cannot rotate lack of variation in the shape of the ankle mortice makes it unable to accommodate the wider diameter of the anterior part of the talus. Rarely cross-union occurs with the fibula so rotated that in plantar flexion of the ankle, the narrower posterior part of the talus is a loose fit in the mortice and the ankle is unstable in that position.

Treatment in plaster

Fractures of the dense compact bone of the tibial shaft are characteristically slow to unite. Nevertheless there are still many fractures of the tibia and fibula which can be treated in plaster. These include the moderately undisplaced fractures or those which can be manipulated to a stable reduction. Other less stable fractures need plaster when blistering skin trouble or severe general illness make open operation impossible.

Plaster does not immobilize a fracture but when applied to the surface of the limb it can often reduce movement sufficiently to allow union although rotation and angulation can occur. The further the plaster is from the bone the poorer its fixation, so that the unpadded plaster is to be preferred. Most limbs waste in plaster and the obese limb in particular needs constant observation. Progressive loosening may demand repeated replastering.

It is often believed that plaster extended above the partly flexed knee prevents rotation but this is not so. The tendency of the lower part of the tibia to rotate is reduced but with the knee partly flexed the upper tibia can rotate on the femur so that movement occurs at the fracture. Fractures of the tibia do better in long plasters with the knee straight and quadriceps exercises are more easily performed.

When the tibia is fractured without injury to the fibula, there is usually good bony contact and union progresses satisfactorily in plaster the intact fibula acting as a splint. If the tibial fracture is displaced manipulative reduction may be difficult and open reduction necessary.

Isolated fractures of the fibula rarely require immobilization and open reduction is almost unknown. The fibula is a hard bone with very little cancellous tissue, union is slow and the fracture may not show sound healing for well over 3 months.

Open reduction and internal fixation

Apart from open fractures operation is indicated (1) when the tibial fracture is likely to be unstable (2) when there is evidence of severe soft tissue damage so that prolonged immobilization in plaster would be detrimental to muscles and joints (3) when there are two or more fractures in the tibia or (4) when other damage to the limb can be treated better with the tibial injury stabilized. Above all

fractures which cannot be reduced by closed manipulation need open reduction. Once the fracture is reduced, advantage should be taken of the exposure to carry out rigid fixation.

Unstable fractures include transverse ones with a large or detached butterfly fragment, most comminuted fractures, long oblique or long spiral fractures which slide about during handling of the limb and most double or multiple fractures. Some spiral fractures are irreducible by closed means owing to a spike of bone being entangled in soft tissues. Other irreducible fractures are the displaced tibial fracture with an intact fibula, the transverse fracture with an interposed fragment of compact bone and the fracture with interposed soft tissue indicated by one fragment sliding about on the other during manipulation without bony crepitus.

The tibia lends itself readily to plating, but the plate must be of adequate length and strong enough to resist angulation (see Chapter 13). Plate and screws must be of similar metal and unlikely to corrode. The screws should be threaded firmly into both cortices. In some spiral fractures two screws will give good fixation if the fracture is accurately reduced. Butterfly fragments may require screwing to main fragments before plating. Other fractures may demand a transfixion screw or cerclage wiring as well as plating. A few spiral fractures with longitudinal splitting may be satisfactorily fixed by wiring alone. No fracture of the tibia is adequately fixed by a solitary screw or single wire.

The intramedullary nail has a place in the treatment of fractures of the tibia. Its insertion requires a small exposure and it can be used with very little disturbance of the soft tissues around the fracture site. The disadvantages are that rigid fixation is more difficult to achieve and the fragments may be held apart by the nail. Böhrer (1957) has forbidden its use for tibial fractures!

Fractures of the tibia and fibula in children

Greenstick fractures of the tibia and fibula are uncommon in children when they do occur some require manipulation. Open reduction of complete fractures with displacement is rarely necessary but occasionally a fracture is not reducible by manipulation and open operation is required.

The common fracture in children is the spiral fracture in the lower half of the tibia with the fibula intact. The child lies with the limb in external rotation, knee flexed, foot in equinus. Often there is no displacement requiring correction. If the knee is allowed to remain flexed, a below knee plaster can be applied with the foot still in equinus. When the cast has set, the knee can be gently extended and a full-length cast completed. No attempt should be made to correct the equinus position of the foot, as there is a strong possibility that the fracture line will open up in its posterior and superior part. Owing to the spiral nature of the bony damage displacement can then occur. At the age at which this injury occurs the tibia is straight so that neither inversion of the foot nor moulding of the plaster should be attempted in an effort to produce the bowing seen in the adult. Angulation of the fracture can be produced by bringing the foot up to a right angle, inverting the foot and moulding the plaster. Whilst many surgeons object to the equinus position of the foot, the normal joints of a child do not become stiff as a result of immobilization and the "abnormal" position is soon corrected by active use once the plaster is removed. The disadvantage of the equinus foot is that the child cannot bear weight in plaster and has to use crutches.

Fractures of the femur

Even in adults most fractures of the shaft of the femur can still be satisfactorily treated by conservative means and the Thomas splint remains most useful. Manipulation may be required to correct displacement whilst traction is necessary to maintain length and prevent redisplacement. Traction can of itself correct displacement in oblique and spiral fractures and is then necessary to maintain length during union. The Thomas splint lends itself to the addition of slings, pads and rigid fulcra which can restore and retain the normal curvature of the femur and resist any tendency to angulation. A hinged knee piece makes it possible to correct forward angulation of supracondylar fractures and can be valuable not only in correcting rotational displacement but also in assisting in the elimination of angulation in the transverse plane. The Thomas splint can be suspended by weights and pulleys so that the limb floats as the patient moves about in bed, thus preventing pain and the tendency for angulation at the fracture to occur during movement.

The closed fracture of the femoral shaft is associated with free bleeding into the soft tissues and one or two litres of blood may be lost. If the dense inelastic fascial sheath of the limb is intact the increase in volume is accommodated by the limb becoming more spherical in shape so that shortening occurs with overlap of the bone ends. Traction is valuable for as the blood is absorbed the shortening and bony overlap may be gradually corrected (Fig. 27).

Open reduction and internal fixation

Operation may be required for closed bony injuries (1) when it is desirable to restore full length and alignment, (2) in comminuted fractures, and (3) in fractures where manipulative reduction has failed or interposed soft tissue makes reduction impossible.

Up to the present a plate sufficiently rigid to secure internal fixation of the femur without supplementary splintage has not been devised.

Intramedullary nailing is probably more used in the treatment of femoral fractures than in any others. Although a little rotational movement and angulation can still occur, the mobility rarely prevents union and splintage after operation is not often required. Restoration of function is usually rapid. The use of the intramedullary nail is most satisfactory for fractures above mid-shaft, including the high spiral fracture with a sharp spike on the upper fragment buried in the anterior thigh muscles (Fig. 28).

When the fracture is below mid-shaft the narrow part of the medullary cavity below the fracture is usually insufficiently long to grip the nail. The nail will then lie in a trumpet-shaped cavity filled with spongy bone which collapses under pressure and leaves the lower part of the femur free to hinge at the fracture unless the nail can be made to penetrate the compact bone of the intercondylar notch or to impinge on compact bone elsewhere in the lower end of the femur. However, if external splintage is also used the intramedullary nail can maintain end-to-end position (Fig. 29).

Intramedullary nails are of great value in treating some bilateral femoral-shaft fractures and where multiple fractures in a limb can be better handled with the femoral injury controlled.

The supracondylar fracture is usually associated with backward displacement of the lower fragment. Sometimes this can be corrected by manipulation with the

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knee flexed. After reduction position can be maintained on a Thomas splint with knee flexion attachment and skeletal traction via the tibial tuberosity. When such manipulation fails, *crossed flexible intramedullary nails* can be introduced after open reduction. The same nails can be inserted via stab wounds, after successful closed reduction they are inserted just above each of the condyles and driven upwards crossing before entering the narrow medullary cavity. Fixation is usually sufficient for early active movements but splintage may be required for a time.

Comminuted spiral fractures between the mid shaft and the supracondylar region common in middle-aged and elderly females may be difficult to reduce and slow to unite. When operative reduction is indicated fixation can be achieved by plating or by a combination of transfixation screws and circlage wires. Splintage is required for at least a month after operation but union is often insufficiently

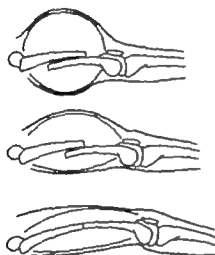


FIG. 27—Haemorrhage into the dense fascial sheath of the thigh causes overlap of a shaft fracture (after Charnley 1950)

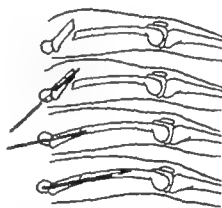


FIG. 28—Intramedullary nailing for a fracture above mid-shaft of the femur gives a firm three point fixation.

sound for weight bearing for at least 3 months. After removal of the splint, movement can be encouraged the patient can sit out of bed and in many instances can be mobilized on crutches. A weight relieving caliper can be provided when early ambulation is particularly desired.

Comminuted and spiral fractures of the shaft are occasionally seen in heavy elderly women with arthritic changes in the hip and sometimes in the knee. These patients tend to be inert and bedsores are the inevitable result. Intramedullary nailing combined at times with circlage wiring can give a satisfactory degree of fixation combined with suspended splintage this will abolish the pain and allow the patient to move about so that pressure sores can be prevented.

Most operations on the femur are followed by an effusion into the knee this subsides rapidly without special treatment and does not seem to contribute to stiffness of the joint.

Plating, wiring and screwing of the femur require supplementary splintage but traction is not required except to maintain the position of the limb on a Thomas

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splint. Internal fixation usually means union in good position and in compatible with active movements earlier than when treatment is conservative.

Fractures of the femur in children

Femoral fractures in children usually present little difficulty in splintage or union. Shortening of one half to one inch is rapidly overcome by an increased rate of growth while angulation is corrected by growth and remodelling. Once a child has walked bowing develops in two planes and it is debatable whether a gallows should be used. The Thomas splint allows the use of pads and slings to maintain the normal curvatures and can be used for children of all ages. For



FIG. 29—Intramedullary nailing of a fracture below mid-shaft of the femur with evidence of movement and compression of cancellous bone (Mr P. S. London's case).

example, a recently born child with a birth fracture of the femoral shaft was successfully treated at home on a specially-ordered Thomas splint attending hospital as an out-patient.

Children settle down on Thomas splints more quickly than on gallows. They are happier; they can move about more freely in bed and can see more of their surroundings. In winter the infant on a gallows can be thoroughly miserable with cold, wet buttocks.

Occasionally in the child a spiral fracture of the middle third of the shaft comes to lie in good alignment, without shortening but with periosteal surface to periosteal surface. One such fracture may unite readily; another may unite poorly with irregular callus and will refracture easily; a third may not unite at all. Poor union and failure to unite are indications for open reduction and internal fixation. At operation it can be seen that the apposing surfaces are covered by more or less intact periosteum from which adequate callus has been unable to form (Fig. 30).

The majority of femoral fractures in children are united in 5-6 weeks. Within



(a)



(b)



(c)

FIG 30—(a) Fracture of the femoral shaft in a child of 11 years (b) unsound union with periosteum-to-periosteum position after 10 weeks (c) good union 9 weeks after open reduction.

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days of removal of splintage mobility is returning and they are eager to walk. Weight bearing can then safely be permitted.

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In the upper extremity mobility is more important than length whilst stability is less important than in the lower limb. Length is concerned with reach and some inches of shortening in the arm can be compensated for by movements of the trunk by extending the legs, or by standing on the toes. Mobility on the other hand, is essential for the patient's care of himself and for the wide range of skills developed by human beings in their occupations, sports and pastimes and for social activities. Full movements require mobility of all joints and between the scapula and the chest wall. Thus the treatment of upper limb fractures must aim to maintain this mobility. Fortunately impaired movements at one joint can be compensated for by movements at others.

In upper limb fractures a degree of consolidation which would be unacceptable in the weight bearing lower limb is often consistent with safe use.

Union with deformity in the arm is less likely to result in adverse joint changes from static causes. Only when deformity or shortening interferes with pronation or supination of the forearm is disability likely to be significant.

Nevertheless, in some heavy occupations strength and stability are all important and sometimes may need to be secured at the expense of full movement.

Fractures of the humerus

Most fractures of the humeral shaft can be treated conservatively. The widely advised hanging cast is not easy to apply, its application is uncomfortable and the position achieved often disappointing. With the movement and leverage that the hanging cast can exert on the fracture it may well be a potent source of non union.

Bandaging the arm to the side with domette, with a pad of cotton wool between the limb and the chest wall, is easily carried out and does not cause pain provided that the elbow is flexed to 90 degrees or less. Three overlapping plaster slabs are next applied from acromion process to elbow anteriorly, laterally and posteriorly outside the domette bandage, to which they adhere firmly. (In thin patients a pad of adhesive felt may be needed over the shoulder.) As the plaster sets it is moulded to fit snugly around the arm. Finally the forearm is supported in a sling with the elbow above a right-angle. Once the patient has learnt to relax he is comfortable and usually the fracture is in good position. The radiograph may show a little backward angulation which can be rectified by increasing the elbow flexion. Patients treated by this method cannot lie flat but need to sleep in a sitting or semi-recumbent position for the first fortnight (Fig. 31).

This plaster shield, which supports and protects the fracture, is required for about a month. Should the plaster become loose, it can be supplemented by further bandaging or the addition of an adhesive bandage. Once there is evidence of healing the bandage and plaster can be removed to permit rehabilitation.

Open reduction

When union of a humeral shaft in good position is particularly desirable and reduction cannot be achieved by closed means open reduction is indicated and

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(a)



(b)

FIG 31—(a) Fracture of the shaft of the humerus in a hanging cast, with deformity (b) end-to-end position of fracture with good alignment in plaster shield.



Fig. 31 cont —(c) The plaster shield

plating is worth while a major indication is when one of the main fragments has buttonholed adjoining muscles or when interposed soft tissue makes the reduction unstable

Distraction following marked soft tissue damage can result in delayed union or non-union and is an indication for operative interference This may also be needed for delayed union or pseudo-arthritis from other causes

Internal fixation may be desired when other injuries to the limb can be better handled with the fracture fixed Satisfactory fixation can be achieved by intra medullary nailing if end to-end reduction can be obtained Only a stab wound is required for the introduction of the nail, the use of which is sometimes advisable for cosmetic reasons Some elderly patients benefit by internal fixation to allow earlier active use Humeral-shaft fractures in the child are usually complete and can be treated similarly to those in the adult Non union does not occur and angulation is corrected by growth

Fractures of the forearm

Many fractures of both bones of the forearm can be reduced by manipulation under radiological control and immobilized in plaster Good reduction is aided by attention to rotational deformity (Evans 1951) Union in the adult is often slow and much supervision is required to detect and correct later displacement. Plaster immobilization is only approximate Wasting of fat and muscle can result in the plaster becoming loose and allowing movement Stiffness develops during prolonged enclosure in plaster with the elbow flexed especially when the forearm is supinated Restoration of function is slow but many make a good recovery

Open reduction and internal fixation

Operation with accurate reduction and rigid plating allows freedom from plaster immediately or soon afterwards Muscle weakness and joint stiffness are reduced The antirotational plate of Hicks (see Chapter 13) is particularly valuable for fractures of the radius

Both bones should be subjected to internal fixation. To plate the radius and to leave the ulna may give a disappointing result. Even in plaster with the forearm in a theoretically correct rotational position depending on the level of the radial fracture muscles act against new resistances and displace the ulna. Although normally the ulna cannot rotate, in these circumstances rotation can occur. Movement can occur at the radial fracture even in plaster when both bones have been plated if the plating has been insufficiently rigid or cannot resist rotation. Non union can then result. Replacement of the inefficient radial fixation by rigid plating even without removing the disc of fibrous tissue between the bone ends can lead to bony union (Fig. 32).

Intramedullary nailing cannot usually be recommended for forearm bones. In the radius such nailing cannot resist rotation, the force most likely to be disruptive to the radial fracture.

Smith (1959) reviewed the results of plating fractures of the forearm bones in the Birmingham Accident Hospital and found that a two weeks delay before operating apparently gave better results than early fixation. This unexpected finding can be partly explained by the fact that emergency operations were carried out on the worst injuries. In some cases the delay before surgery could have permitted progressive improvement of a poor blood supply which might have been completely cut off by early operation.

Provided that a large amount of callus has not formed fractures of the radius and ulna 2-3 months old can be successfully treated by open reduction and internal fixation. More care is necessary at operation for the bones are softer. Shortening of soft tissues may necessitate step-cutting to allow apposition of the bone ends without force which could damage the softened bone. Careful plating should follow. Late operation usually requires a plaster cast for a time afterwards.

Apart from Galeazzi fractures, which may show a separation of the ulnar styloid, fractures of the radius with an intact ulna are uncommon. Such injuries can be treated by closed manipulation and plaster but restoration of function is quicker and more complete if rigid plating is used.

Fractures of the ulna alone treated in plaster usually unite without difficulty but with an excess of callus. The excess of callus indicates mobility at the fracture site probably of a rotational type. Nevertheless, the patient is comfortable in such a plaster union occurs in a reasonable time, and the excess of callus does not seem to be detrimental.

Plating without plaster is needed for non union and may be desirable when the fracture is angulated and manipulation fails to reduce it.

Fractures of the forearm in children

There is a great variety of greenstick fractures of the forearm. If the shaft of the radius is involved the ulnar styloid may be fractured or the lower ulnar epiphysis displaced. When the injury involves both shafts they are usually broken at different levels with similar angulation. When the fractures are at the same levels the radius is more angulated than the ulna.

Most greenstick fractures are described as having been caused by a fall on the outstretched hand. The causative violence is a combination of longitudinal compression and torsion except in rare instances when both bones are broken at the

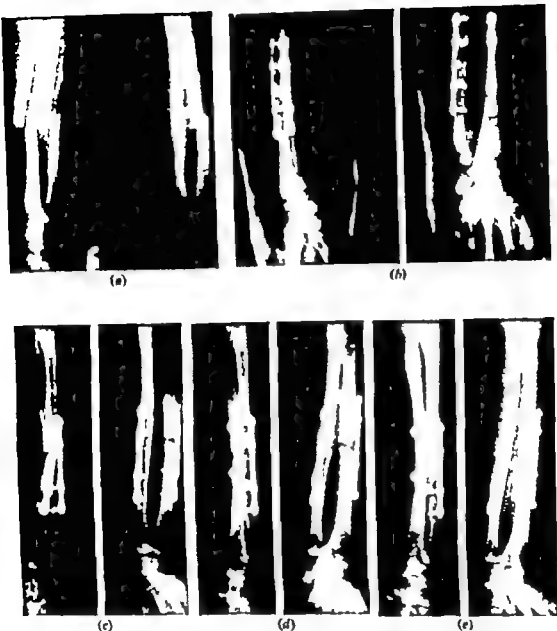


FIG. 32.—(a) Fracture of radius and ulna (b) position after plating on the day of injury (c) union of the ulna and fibrous union of the radius 53 weeks after injury (d) radius rigidly plated fibrous union undisturbed (e) sound union of radius 16 weeks after rigid fixation.

same level and equally angulated from a true angulating force. In the fall the hand in contact with the ground fixes the lower end of the radius and ulna with the body weight swinging so as to tend to pronate the forearm the radius is twisted beyond its limit of elasticity and breaks. The upper part of the bone tends to continue to pronate while the lower part remains relatively supinated so that backward angulation occurs. Once the radius has broken, the diminished violence fractures the ulnar styloid or produces a greenstick fracture of the ulna with less angular deformity which may as a result of the tensile property of the immature

bone, undergo almost complete spontaneous reduction. In a few instances the inferior radio-ulnar joint seems to absorb the remaining violence.

The degree of angulation depends on the momentum imparted to the limb by the swinging body weight. The level of the fracture may depend on the degree of dorsiflexion of the wrist as the hand strikes the ground, probably the greater the dorsiflexion the lower the level of the radial fracture. Almost without exception the ulnar fracture is lower than the radial.

Not uncommonly the radius is the site of a complete fracture with the lower fragment lying behind the upper and with overlap. The fracture is always in the lower third of the bone.

When the body weight swings in the opposite direction the upper part of the radius tends to supinate while the lower fragment remains relatively pronated resulting in a fracture with forward angulation.

Lesser angulations need no correction as they will correct themselves in time. The injured limb is better protected by plaster to prevent further injury or increase in deformity.

In the correction of angulation methods of manipulation advocated have included the completion of greenstick fractures and direct pressure over the prominence or leverage over a padded wedge. The first manoeuvre cannot be recommended because it produces fractures that are unstable and often defy manipulative reduction. The intact periosteum is torn across and further damage to the blood supply shows itself in slower union. The second and third methods produce a temporary correction of deformity which recurs in plaster. They are applicable only to the rare greenstick fractures resulting from a pure bending force.

Reduction of the majority of greenstick fractures is easily carried out by rotating the limb in the direction opposite to that which produced the deformity. The limb is grasped above and below the injury and twisted so as to "unscrew" the deformity.

Backward angulation fractures (supination deformities) require plaster immobilization with the forearm in full pronation and with a little flexion of the wrist. The manipulation aligns the relatively supinated lower fragment with the relatively pronated upper part of the radius and renders taut the intact soft tissues—the hinge—on the posterior aspect of the injury. These soft tissues are not damaged by this manipulation. While the hinge remains tightened it resists the tendency of muscle pull to bring about redisplacement. When angulation is marked the plaster will need extension above the flexed elbow; this will maintain full pronation for the period required for adequate union, usually 4–6 weeks. Children so treated do not develop stiff joints; impaired movement on removal of the plaster is soon overcome by active use. There is no need to bring the forearm back to the mid-rotational position before application of the plaster; otherwise the hinge is relaxed and partial or complete redisplacement is permitted. Nor is there any need to apply fresh plaster with less pronation after 2 weeks. Removal of the above-elbow part of the cast after 2–3 weeks is unnecessary. Radiographically the fracture may appear to be united after 3 weeks. Removal of plaster at this time from limbs not subjected to manipulation is desirable. As regards the more severely injured bones, although the fracture may apparently be united, too early removal of plaster is followed by a number of so-called refractures.

Manipulation for correction of forward angulation, the pronation deformity, aligns the lower pronated part of the radius with the upper supinated fragment.

THE UPPER LIMB

The hinge situated anteriorly becomes taut and plaster is required in full supination with the wrist in partial dorsiflexion. According to need the plaster may or may not be extended above the elbow.

When the fracture of the radius is complete and radiography shows overlap traction is essential before the fragments can be placed end-to-end. Continuous traction for some hours may be necessary to overcome the shortening if the injury is more than a few hours old and there is much swelling. As there is no soft tissue hinge many of these fractures tend to angulate backwards even in plaster.

Redisplacement is favoured by buckling of the posterior cortical bone adjoining the fracture surface on the lower fragment. The deformity may require further manipulation but an angulation of 20 degrees or less will be naturally corrected in most of these fractures which are so commonly seen in children under 11 years of age.

REFERENCES

- Böhler L. (1938) *Technik der Knochenbruchbehandlung*. Vienna: Maudrich.
— (1957). Bericht über die bei 3308 Unterschenkelbrüchen in den Jahren 1926-1950 im Wiener Unfallkrankenhaus erzielten Behandlungsergebnisse unter Benützung des Hülkerlithersverfahrens. Berlin: Springer Verlag.
Charnley J. (1950). *The Closed Treatment of Common Fractures*. Edinburgh: Livingstone.
— (1953). *Compression Arthrodesis*. Edinburgh: Livingstone.
Danis, R. (1949). *Théorie et Pratique de L'ostéosynthèse*. Paris: Masson et Cie.
Evans, E. M. (1951) *J Bone Jt Surg.*, 33B: 548.
Report: Chief Medical Officer of the Ministry of Education (1958). *Fifty years of the School Medical Service: The Health of the School Child*. London: H.M. Stationery Office.
Smith, J. E. M. (1959). *J Bone Jt Surg.*, 41B: 122.

CHAPTER 11

FRACTURES NEAR OR INVOLVING JOINTS OF THE UPPER LIMB

INTRODUCTION

F G BADGER

FRACTURES near to or involving a joint vary from trivial fissure fractures with no subsequent problems of union or joint stiffness to severe joint damage complicated by major injury of adjacent long bone or soft tissue. Capsular and ligamentous injury may be minimal or the fracture may be of minor character the ligamentous injury severe. Damage to bone and cartilage may be of overriding importance or the joint may be involved as only part of a grossly injured limb. In treatment, restoration of function is the chief objective but the correction of alignment may be equally important.

Significant joint trauma will produce a haemarthrosis to which impaired mobility has often been attributed. The good results that follow arthrotomy for intra-articular lesions indicate that a small or moderate collection of blood in a joint readily undergoes absorption and therefore that the haemarthrosis cannot itself be regarded as the cause of the stiffness which so often follows joint injury. Only after repeated haemorrhages does the joint of a haemophilic begin to show a diminished range of movements. Thus the routine removal of blood from the injured joint is not indicated.

If an articular fracture is unstable it is probable that movement provokes recurrent damage and repeated bleeding into the joint which can then become the site of adhesions but the bone is not the only structure involved—joint injuries with any degree of subluxation or dislocation are associated with far more damage to capsule, synovium and ligaments than is evident from the radiological appearance. In the shoulder and elbow particularly such damage often extends into adjacent muscles. In addition a whole segment of the limb including uninjured joint structures, is involved in an inflammatory reaction. Unresolved tissue oedema can lead to localized or diffuse fibrosis. Irritation of the zone of inflammation by ill-advised movement may prolong the reaction and increase fibrosis in all or any of the surrounding soft tissues. Elasticity becomes lost, muscles shortened, ligaments thickened and adhesions develop.

Perkins (1953) maintained that the inflammation due to injury ceases quickly. He recommended rest during the period of inflammation and activity during repair regardless of the fact that too early mobilization can break down granulation tissue causing bleeding and further reaction.

Specific injuries to muscles, tendons and ligaments can cause shortening or abnormal attachments thus limiting movement. Damage to cartilage and bone may lead to the development of arthritis with pain and stiffness. Finally new bone formed during healing may limit movement by producing a mechanical block.

PRINCIPLES OF TREATMENT

PRINCIPLES OF TREATMENT

Treatment is aimed at combining the optimal conditions for healing of tissues in the best possible position with reasonably rapid restoration of function

When the bone lesion is minimal there is no danger of displacement and union will be certain. The question of fixation or movement depends on the nature and extent of the soft tissue injury and the particular joint involved. Rupture of major ligaments is best treated by immediate repair followed by a suitable period of rest in plaster.

When the bone lesion is significant but the displacement slight a period of immobilization may be necessary to allow the soft tissues to heal. When the articular surfaces are involved in a bony lesion with displacement accurate reduction is important. If it cannot be easily secured by closed manipulation and splintage operative intervention is advisable provided that it is likely to succeed.

Bony fixation usually demands the introduction of metal with or without bone grafting, although bone pegs have been used. Metal near a joint has been described as "dangerous" and blamed for joint stiffness. In fact metal is safe provided that corrosion does not occur although the presence of even a safe metal may contribute to the production of joint stiffness should infection supervene.

Other causes of loss of movement after a joint fracture are (1) the severity of the original injury (2) operative trauma aggravated by rough handling, a wrongly placed exposure, and failure to achieve a stable reduction (3) irreparable damage to extra articular structures (4) too early mobilization or (5) unduly prolonged fixation.

When reduction is impossible or cannot be maintained early movement is advised in the hope of bringing about a smoothing off of the opposing joint surfaces, while maintaining the power of muscles and elasticity of ligaments. Opinions differ more on the indications for such "active" non-intervention than on any other aspect of the treatment of injuries.

In a few instances early arthroplasty sometimes using a metallic or plastic prosthesis, may be considered. rarely immediate arthrodesis is advisable and possible. Later arthrodesis or less commonly arthroplasty may be needed if arthritic changes supervene. Occasionally some form of ligamentous replacement may be advised.

DIGITS

Dislocation of interphalangeal joints are common and frequently radiological examination after reduction shows a chip fracture which was not seen in the original film. Mostly these injuries involve the anterior margin of the base of the middle phalanx and the bony fragments are avulsed by one of the attachments of the flexor digitorum sublimis tendon.

When the displacement is slight healing occurs with splintage or plaster. Wide separation suggests a need for operation. The bony fragments can be replaced and held by soft tissue suture or excised and the tendon reattached. At operation widespread damage can be seen involving joint capsule and tendon sheath. Following repair healing is usually rapid and restoration of function gratifying after removal of plaster in 3-4 weeks. Without operation, swelling may persist for many weeks and flexion is often permanently limited.

Other fracture-dislocations in the fingers may be associated with splitting of the base of a phalanx or separation of part of a phalangeal head. Such fractures may benefit from open reduction and pegging into place with Kirschner wire. Replacement and fixation of the bony fragment sometimes seen in mallet fingers can be worth while. Avulsion fractures also occur at the bases of the proximal phalanges. Operative reduction and fixation is particularly indicated in the thumb.

Carpometacarpal Joints

Fracture-dislocation of the first carpometacarpal joint (Bennett's fracture) has been treated in various ways. A supporting bandage and early use has been recommended as an alternative to manipulation and plaster with or without traction. Moulding of plaster to maintain the reduction of the dislocation has often led to sloughing of skin. All the fractures treated by either of these methods united mostly with displacement. Despite favourable accounts of the results of such treatment a critical survey of patients so treated will reveal some loss of movement at the carpometacarpal joint, weakness of the thumb and often pain. Early operation for reduction and fixation by screwing will give bony stability. Plaster is not needed; painless movement is rapidly restored and the fracture unites without difficulty (Badger, 1956). Screwing is applicable only to the larger bony fragments. The smaller ones still need to be handled conservatively. Occasionally a fracture-dislocation of the fifth carpometacarpal joint requires a similar operative repair (Fig. 33).

CARPUS

The scaphoid is the carpal bone most commonly fractured; the majority of such injuries heal without difficulty if the wrist is held in plaster for a suitable period. Delayed union or non union may be an indication for operative interference. Screwing has been advocated and bone grafting successfully carried out (Thomas, 1949).

Patients with old-established non-union of the scaphoid may be handicapped by pain and increasing stiffness from osteoarthritic changes in the radiocarpal joint. The pain can be relieved by arthrodesis but this does not help the patient who needs a mobile wrist. Excision of the scaphoid may alleviate the pain but results in weakness of the pincer hold between the thumb and index finger. Replacement of the scaphoid by a plastic prosthesis has been advocated but a successful series of cases has not been published.

Excision of the radial styloid with the degenerate part of the lower articular surface and proximal part of the scaphoid eradicates the osteoarthritic part of the radiocarpal joint. The opposing surfaces of the distal fragment of the scaphoid and the capitate can be denuded of cartilage and the resulting space filled in with a bone graft cut from the excised radial styloid and held in place by a wire loop. Successful fusion of the remains of the scaphoid to the capitate results in a painless mobile and moderately strong wrist. The author has carried out this operation successfully on 8 patients including a pipe fitter and a glass blower (Fig. 34).

WRIST

There are several types of fracture-dislocation of the wrist: perilunar, perilunar trans-scaphoid, and perilunar trans-scaphoid with fracture of radial styloid or

WRIST

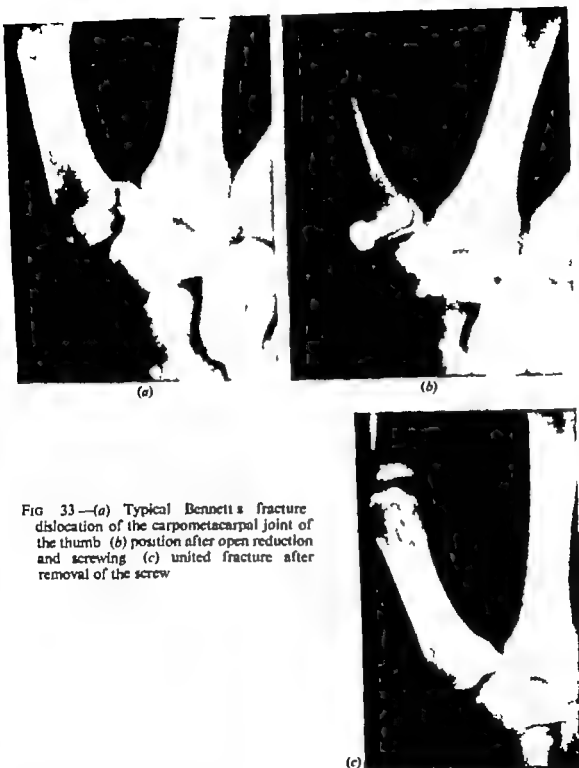


FIG 33—(a) Typical Bennett's fracture dislocation of the carpometacarpal joint of the thumb (b) position after open reduction and screwing (c) united fracture after removal of the screw

posterior margin of radius. Manipulative reduction can be successful but may need to be supplemented by thumb traction. Except in perilunar dislocations plaster is required for a long period and even then there may be non union of the scaphoid. These injuries to the wrist are associated with widespread ligamentous injury. Conservative treatment is usually followed by stiffness and sometimes pain even



(a)



(b)



(c)

FIG 34 —(a) An old fracture of scaphoid with established non-union and osteoarthritis of the radiocarpal joint (b) appearance after excision of proximal pole of scaphoid, radial styloid and arthritic part of articular surface of the radius bone graft inserted between scaphoid and capitate position held by wire suture (c) sound fusion of the scaphoid to capitate wrist is mobile and painless.

when the scaphoid has united. Operative repair of the ligaments makes reduction stable and its retention in plaster simpler. The resulting function is better than with conservative treatment.

Anterior marginal fractures of the radius with forward and upward subluxation of the carpus can be treated by manipulation and plaster with thumb traction.

FOREARM

Some of these injuries are amenable to open reduction and fixation by screwing. Plaster is required for a few weeks but the discomfort produced by traction on the thumb is avoided.

FOREARM

There are two fractures of the forearm associated with distant joint injuries (1) the Galeazzi fracture with a fracture of the radial shaft and a dislocation of the inferior radio-ulnar joint and (2) the Monteggia fracture with dislocation of the superior radio-ulnar joint and a fracture of the ulna.

Galeazzi fracture

In the Galeazzi injury a somewhat oblique fracture is situated at the junction of the middle and lower thirds of the radius. The soft tissue injury extends from the ulnar styloid which may be fractured through the dislocated inferior radio-ulnar joint and up the interosseous membrane to the fracture of the radius; the soft tissues are stripped from the radius below the fracture on the medial aspect and above the fracture on the lateral surface with an oblique tear at the fracture line. The injury is caused by a combination of longitudinal compression and forced pronation. It is often unstable, but may be reducible by traction and supination. Reduction can be held in plaster with the limb in full supination but union is slow with resulting stiffness and impaired pronation. Often internal fixation is needed but non union has been relatively frequent with bony absorption and further deformity sometimes leading to further operations and some deplorable end results. The cause is not completely known but the soft tissue injury combined with an orthodox exposure of the radius may result in an impaired blood supply to the bone with slow healing. When, in addition, the plating has permitted movement a bad result is inevitable. Better results can be achieved by rigid plating, using an incision which takes advantage of the bony stripping caused by the injury. The plate must resist rotation which intramedullary nailing cannot do effectively.

Monteggia fracture

The Monteggia fracture, as Evans (1949) has shown, can be due to forced pronation but the injury can also be caused by direct violence as in protecting the head from a blow. Such an injury may not be reducible by the manipulation described by Evans. Open reduction and plating of the ulna may be needed and in some instances instability of the reduction of the radial head injury may necessitate repair of the torn orbicular ligament.

The so-called "flexion Monteggia fracture" may be regarded as a variant of the dislocation of the elbow. The injury is characterized by backward dislocation of the radial head, which may be fractured and an unstable angulated fracture of the ulna. It may be reduced by manipulation but needs prolonged immobilization in plaster with the elbow extended. Union is slow and followed by stiffness of elbow and forearm. Plating the ulna is often essential but open treatment of the radial head fracture should be avoided until the ulna is soundly united. Early operation on the radial head contributes little or nothing to the restoration of function.

ELBOW

Three bones articulate at the elbow which is the site of many fractures and fracture dislocations

Radial head

A fracture of the radial head may present as a painful wrist and the inexperienced surgeon may be at a loss to explain the absence of radiological bony injury in spite of a history of a fall on the outstretched hand. The pain is produced by rotational movements of the forearm and is presumably referred along the sensory branch of the radial nerve which gives a twig to the radiohumeral joint. Other injuries to this joint can be associated with similar referred pain. When the radial head is fractured, there may well be local tenderness. Flexion and extension of the elbow may be painful and there may be a small effusion into the elbow joint. The more comminuted the radial head the greater may be the pain in the wrist.

Except when associated with a dislocation of the elbow or other bony injuries, fractures of the radial head are due to a combination of longitudinal compression and abduction violence applied to the extended elbow. While the radiograph shows the bony damage it does not reveal the co-existing soft tissue damage—a tear of the medial ligament and often of the common flexor origin. The physical signs of this associated injury are easily overlooked or their significance misunderstood. Healing of this part of the injury will be quicker and associated with less fibrosis if the torn tissues are allowed to lie in apposition and remain at rest. Immobilization in plaster is to be preferred to early active movement.

The greater the violence the more the comminution of the radial head and the more extensive the soft tissue injury. Immediate or early excision of the radial head is often practised but operation damages the remaining intact soft tissues on the outer side of the joint. If early active movements are prescribed, neither wound is at rest, healing is slow and repair takes place with an excess of scar tissue. Eventually there is some movement at the elbow and the operator may believe it to be better than if he had not intervened. At operation it may be found that the widely displaced fragments of the radial head had been projected through the capsule into muscles by almost explosive violence. Being extra articular they constituted no source of obstruction to movement.

Replacement of the radial head by a plastic prosthesis has been practised but the results were disappointing in both early and delayed operations. Many fractures of the radial head can be rested in plaster for 3 weeks to the benefit of the patient. Easy active movements and exercises alternated with a rest in a sling can follow. After a week the sling can be discarded. No pain should be caused by movement. Complete radiological healing of the fracture may take 3 months or longer.

Once the elbow has settled down, excision of the radial head can be carried out if indicated and without aggravating the original injury. Deformed radial heads, however, can give good and painless movement.

Rarely the radial head may be injured with displacement of almost a quadrant but with minimal signs on the inner side of the joint. In a young patient the fracture can be subjected to open reduction and the fragment held by a fine stitch of catgut passed through the bone. The fracture will heal to give a normal radial head (Fig. 35).

ELBOW

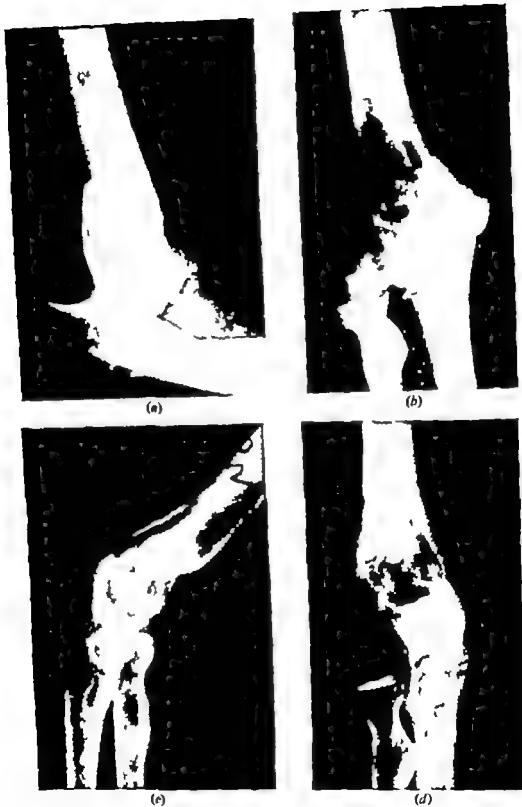


FIG 35—(a b) Dislocation of the elbow dislocation of superior radioulnar joint and fracture of the head of the radius (c d) dislocations reduced, radial head repaired by suture and soundly united.

Fracture-dislocations

All dislocations of the elbow are associated with major ligamentous injuries and some are complicated by a fracture of one or more of the bones including the radial head, coronoid process, medial epicondyle, capitellum and trochlea. Rarely if ever does the fracture of the coronoid need excision but any of the other fractures may require operation. If open treatment is indicated, the operation should be early and the exposure so placed that the joint is entered through the soft tissue tear arising from the injury. After treatment of the bony damage the soft tissues can be sutured. Rest in plaster will promote soft tissue healing.

Medial epicondyle

The medial epicondyle may be avulsed as part of the injury in a dislocation of the elbow especially in children and adolescents. The injury is also seen without apparent dislocation, although during operative treatment it can be shown that there must have been a dislocation or subluxation of the elbow with spontaneous reduction. The associated tearing of soft tissues is comparable with that found when dislocation has been reduced, but is usually less extensive.

The separated epicondyle may be displaced to lie between the ulna and humerus in the dislocated elbow. Manipulation can remove the epicondyle but even after reduction of the dislocation it may still lie between the olecranon and humerus. Further manipulation assisted by faradism may bring about its replacement and it will unite in plaster. Very often the epicondyle lies below its original site and can only heal by fibrosis.

Operation is desirable for those patients with the more marked displacements, whether or not dislocation is known to have been present, and in those in whom the epicondyle has defied attempts to remove it from the joint by manipulation. With the patient lying face down the injury is exposed by means of a medial excision. Preferably the ulnar nerve should be displayed and protected. The displaced piece of bone can be reduced and fixed in a variety of ways. Excision has advantages in that only a soft tissue repair is needed and the possibility of ulnar neuritis due to irregularity of the nerve bed is removed. Transplantation of the nerve is unnecessary. The epicondyle is avulsed from the humerus, leaving a crater to the lip of which the torn ligament and common flexor origin can be attached by a suture passed through a drill hole. The capsule, ligaments and muscles can be further repaired. Active use can be permitted 3 weeks after operation.

Capitellum

In the adult and less commonly in the child, fractures of the capitellum involve separation of the anterior hemispherical part of the articular surface. Part of the trochlea may be attached. The injury may be found without dislocation, and usually the fragment is displaced upwards. Manipulation can be effective and stable, but usually is unsuccessful. Open reduction through a lateral incision can achieve repositioning which can sometimes be retained by soft tissue suture and flexion of the elbow. A wire suture may hold the fragment or a screw can be used. It is introduced just behind the lateral epicondyle and directed forwards and inwards to hold the displaced fragment without penetrating the articular surface. Alternatively the screw can be driven in from the antero-lateral part of the articular surface if

ELBOW

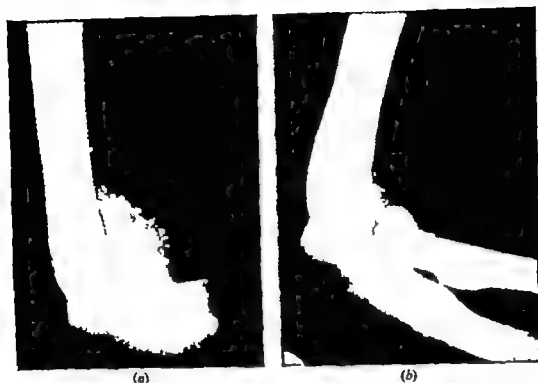


FIG. 36—(a) Fracture of the anterior part of the capitulum (b) position after open reduction and insertion of countersunk screw from antero-lateral part of the articular surface

countersunk so that the head will lie deep to the surface. In older patients excision of the fragment and smoothing down of the raw edges of bone can give a good functional result (Fig. 36)

Trochlea

Displacement fractures of the trochlea are uncommon. They usually demand open reduction and fixation by screwing, using a medial approach.

Olecranon

Fractures of the olecranon are usually the result of a fall on the elbow or a blow over the olecranon which fractures from impact against the lower end of the humerus. The injury varies from a fissure to gross comminution. Occasionally a transverse fracture can be reduced by extending the elbow. Immobilization in extension is usually followed by union but generally with much stiffness, especially in older patients. Operation may aim at bony repair or excision with restoration of the extensor mechanism. The olecranon is repaired by wiring or screwing if the fracture is not comminuted and the joint is not arthritic. A solitary screw from the tip of the olecranon to the dense cortical bone at the base of the coronoid gives better fixation than the longer screw introduced along the medullary cavity of the ulna, and allows early mobilization. Wiring usually needs the support of plaster for a few weeks (Fig. 37).

Excision is reserved for comminuted fractures especially when compound, or transverse fractures with separation in aged patients always provided that the



FIG 37—(a) Fracture of the olecranon (b) position after reduction and screwing.

elbow is stable and forward subluxation of the ulna on the humerus cannot occur. The operation converts the bony injury to a soft tissue injury with quicker healing. Repair of the extensor mechanism can be by soft tissue suture or apposition of the triceps tendon to raw bone by suture through drill holes. Early mobilization can be permitted in the osteoarthritic joint. Occasionally excision of the olecranon may make combing the hair difficult. Loss of bone causes relative shortening of the triceps tendon when the shoulder is elevated tension is produced on the long head of the triceps and flexion of the elbow is thereby restricted.

FRACTURE OF THE LATERAL CONDYLE OF THE HUMERUS IN CHILDREN

The description, fracture of the lateral condyle is not quite adequate as the fracture also involves the trochlea. Two types of fracture occur one where the capitellar centre of ossification is displaced together with what appears to be a thin flake of juxta-epiphyseal bone and a second where the associated bony fragment appears as a triangle (Wilson, 1954 Badger 1954). The first type may appear little displaced and may remain so until healing. Alternatively gross displacement may develop within a week despite plaster immobilization. The injury can be produced by abduction. The second type can also happen as the result of violence transmitted up the radius, punching off a large piece of the humerus. A fall on the olecranon may cause either type the bony ridge of the greater sigmoid notch acting as a wedge to split off the lateral third of the trochlea, capitellum and a variable amount of metaphysis. The injury occurs at an age when the trochlea is entirely cartilaginous so that radiographs reveal only a small part of the injury.

SUPRACONDYLAR FRACTURES OF THE HUMERUS

When displacement can be seen manipulative reduction can be effected but recurrence of deformity is usual. Closed reduction is not easy as the displaced osteo-cartilagenous fragment may be rotated in two planes and held by the pull of the radial extensors of the wrist.

Displacement which cannot be corrected recurs after manipulation or develops in a previously undisplaced fracture requires operation.

At operation the effect of the pull of the attached muscles can be seen also it will be noticed that pressure on the olecranon can force the ridge on its articular surface into the trochlear fracture line, producing redisplacement and explaining how deformity occurs in the initially undisplaced injury. Retention of reduction can usually be effected by suturing the periosteum which is thick and tough. Rarely wiring or screwing is needed. On occasions a similar injury is encountered in adults.

SUPRACONDYLAR FRACTURES OF THE HUMERUS

"Y" and "T" fractures

"Y" and "T" supracondylar fractures of the humerus occur in the adult as a result of a fall on the flexed elbow the upper end of the ulna splitting the condyles asunder. Many of the patients are young and active and fit for operation which is the only means of obtaining full reduction. Exposure by medial and lateral incisions permits reduction and fixation by screwing the condyles together. Attaching the reconstituted lower end of the humerus to the shaft is difficult without increasing the detachment of soft tissues. Until a plate suitable for the purpose has been perfected there is much to be said for aligning the condylar part of the humerus with the shaft by manipulation after closure and holding the position by plaster. With union in reasonable time the functional result is usually good the residual disability being a loss of 10-20 degrees of extension. This is better than that obtained by hanging the limb in a sling without any attempt to improve the position, although such treatment is of value when active intervention is contra indicated. All is not lost when the general or local condition of the patient prevents reduction and a useful range of mobility may be obtained.

Supracondylar fractures in children

The common supracondylar fracture seen in children varies from a fissure to a complete fracture with gross displacement. Intermediate is the greenstick fracture with backward angulation. In the complete fracture with separation the fracture line passes from without inwards and above downwards and forwards, resulting in a sharp antero-medial spike on the shaft fragment the condylar fragment may lie almost at right angles to the shaft postero-laterally.

The causative violence is a fall on the outstretched hand with the elbow incompletely extended and the body weight moving so as to twist the limb laterally whilst the hand is fixed to the ground. After disruption of the bone the humeral shaft continues to move outwards and a little downwards so that the sharp spike on its lower end buries itself in the muscle and deep layers of the skin causing a characteristic patch of bruising just above the elbow anteriorly and a little medial to the mid line. The lower end of the humerus now lies relatively medially rotated while the

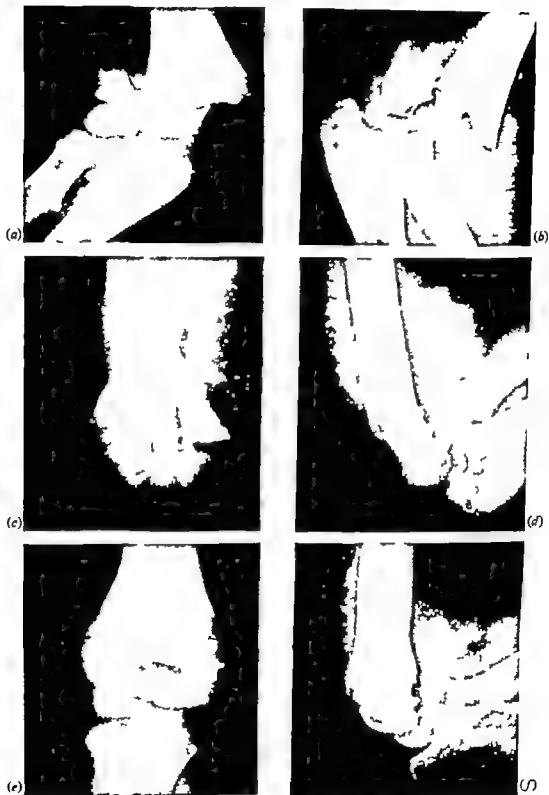


FIG 38 —(a and b) Typical supracondylar fracture of the humerus with severe displacement in a child (c and d) position after manipulation (e and f) union of the fracture in normal alignment

SUPRACONDYLAR FRACTURES OF THE HUMERUS



FIG 38 *cont*—(g) First part of manipulation traction applied with elbow flexed (h) traction continued with grasp on epicondyles (i) manipulation completed



shaft is externally rotated. Rarely the injury is open, when it can be seen that the lower end of the shaft fragment is sharp and chisel-like and may have two important structures stretched over it—the brachial artery and the median nerve.

Reduction by traction on the extended elbow has been advised but since the artery and nerve are fixed by the fascia of the upper forearm and the lower humeral fragment by reason of its position behind the shaft acts as a fulcrum the artery and nerve are stretched by attempted extension and further stretched by traction on the limb. Such a manoeuvre can damage the artery causing spasm and a Volkmann's contracture and injure the median nerve at the same time. Disentangling the bony spike from the soft tissues is difficult when this manoeuvre is attempted.

Manipulative reduction at the earliest possible moment is recommended but traction should be carried out with the elbow flexed and the soft tissues relaxed.

Once the spike is freed from the hole in the muscle the shaft fragment lies in neutral rotation the condylar fragment can be aligned by traction on the epicondyles with one hand and forward pressure with the other during which time the elbow flexes to about 90 degrees. If reduction feels stable a radiological check on position is advisable (Fig. 38).

When the reduction is approved the limb requires a plaster back-slab held by a crepe bandage over cotton wool. Strapping the whole limb to the chest wall is desirable to prevent attempts to use the limb. Such movement could exert a rotational force on the fracture, thus causing the redisplacement so often seen when the limb in plaster is merely allowed to hang in a sling. Occasionally the manipulative reduction fails a stable position cannot be achieved as the lower fragment slides about on the upper. Immediate operation is then required exposure of the fracture through an anterior incision shows that a semicircular flap of dense periosteum torn from the lower part of the shaft has travelled backwards with the condylar fragment so as to lie between the fracture surfaces. Removal of the periosteal flap leads to reduction under vision and the position can be held by suture of the periosteum.

Böhler (1937) described the use of an abduction splint to maintain reduction after manipulative reduction of the supracondylar fracture of the humerus, and stressed the importance of pronating the forearm. This manoeuvre may sometimes be helpful in achieving a good position. Madsen (1955) advocated immobilization in a plaster shoulder spica so as to hold the shoulder in partial abduction and the arm in external rotation with the elbow partly flexed.

A type of supracondylar fracture in some ways resembling the injury seen in children is seen in the aged. The causative injury is similar and produces a posterolateral dislocation in the younger adult. In the child, with the thick articular cartilage, the upper end of the ulna fits well into the coronoid and olecranon fossae, so gripping the lower end of the humerus, and the violence transmitted up the forearm pushes and twists off the lower end of the humerus. In the aged, with loss of depth of articular cartilage and a narrowed joint space, once again the lower end of the humerus is gripped by the ulna and a similar injury can result. Treatment may resemble that required for the injury in the child, but the displacement is usually less.

A fall on the elbow may result in a transverse supracondylar fracture in the child—a greenstick fracture with forward angulation. The deformity may be overlooked unless examination of a true lateral radiograph shows an angle between shaft and capitellum of 90–100 degrees instead of the normal 120–135 degrees. A comparative film of the uninjured elbow confirms the difference in angle and the diagnosis. Treatment is by manipulation and plaster in extension (Fig. 39).

SHOULDER

Excluding the clavicle, the commonest fracture in the shoulder region is that of the surgical neck of the humerus most often seen in the middle years of life and in the aged. In most patients no correction of position is indicated and the shoulder just needs to be put at rest. A sling gives comfort while the patient is sitting quietly but twinges of pain are experienced when moving about. Attempting to lie down at

SUPRACONDYLAR FRACTURES OF THE HUMERUS

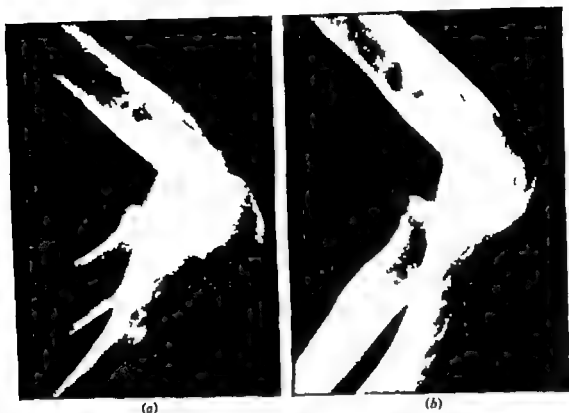


FIG 39—(a) Greenstick supracondylar fracture of humerus with forward angulation
 (b) film of uninjured elbow for comparison
 (c) angulation corrected by manipulation



night can be painful for gravity causes the arm to fall backwards, angulating the fracture. If the arm is bandaged to the side with cotton wool between the limb and the chest wall the patient will be more comfortable. The bandage should be applied

so that the hand can be removed from the sling for active elbow exercises which will prevent cramp. Rebandaging may be necessary but after a short period the bandage can be dispensed with. A sling will be required for another week or so during which time active movements can be started.

In some of the older patients the displacement may be of such a nature as to make manipulation worth while. Once it can be felt that the upper end of the shaft previously in the axilla has been replaced below the humeral head, reduction can be held by abducting the arm across the body over a generous axillary pad. Elastic strapping is used to hold the upper arm in position and the forearm is suspended in a sling with the elbow well flexed. Objection may be raised on the score of constriction of the chest, but the injury occurs in a subject who often already has a more or less immobile chest wall and who breathes with the diaphragm. The strapping does not seem to cause any additional respiratory embarrassment.

Fractures associated with dislocations

Separation of the greater tuberosity of the humerus may occur as an isolated injury but it is usually associated with a fracture of the surgical neck or dislocation of the shoulder.

After reduction of a dislocation by the Kocher method it may be found that the displacement of the tuberosity fragment has increased. For this reason reduction of what is regarded as a straightforward anterior dislocation of the shoulder by the Kocher method without x ray examination and without anaesthesia is to be condemned. Some avow that the additional injury of a fractured tuberosity makes the injury more painful but this is quite unreliable.

Manipulation by an up-to-date version of the method employed by Hugh Owen Thomas achieves reduction of the dislocation and a good position of the fracture. Thomas knew that abducting the arm brought the displaced humeral head down to the rent in the antero-inferior part of the capsule and that traction assisted by pressure in the axilla replaced the humeral head in the glenoid fossa. This manipulation caused the displaced bone to retrace its course without significantly adding to the injury. He used the method without anaesthesia but needed two or more assistants to overcome the muscle spasm resisting the reduction.

Kocher's method made use of leverage to overcome the muscle spasm which rapidly yields nowadays to the use of relaxants. Thus with the aid of an anaesthetist the arm can be abducted, traction applied and the humeral head lifted over the lip of the glenoid by finger and thumb pinching through the anterior axillary fold. The manipulation can be carried out by the surgeon without the aid of assistants.

Fracture of the surgical neck of the humerus complicating dislocation of the shoulder is a severe injury. Although Böhler (1937) claimed to have effected reduction by mechanical traction combined with manipulation, there is much to be said for open reduction carried out as an emergency procedure. In younger patients a bone graft inserted into the humeral head and driven into the medullary cavity of the shaft holds the fracture in the reduced position once the dislocation has been rectified. An intramedullary nail through the greater tuberosity will serve the same purpose.

The injury is also encountered in obese, middle-aged, or elderly women when the humeral head can be palpated in the axilla. Removal of the humeral head through

SHOULDER



FIG. 40 —(a and b) Posterior fracture-dislocation of the shoulder (c and d) dislocation reduced and fracture united after repositioning of bone and insertion of wire mattress-suture. (Cont overleaf)

FRACTURES NEAR OR INVOLVING JOINTS OF THE UPPER LIMB



(e)



(f)



(g)



(h)

FIG. 40 *cont* —(e-h) Range of shoulder movement following healing of injury

an axillary incision gives the best function and greatest comfort in the shortest time. The least common but most troublesome fracture-dislocation is the posterior variety: in this the head is completely detached from the shaft and lies in a pocket on the dorsum of the scapula below the spine, having been driven through a posterior capsular tear close to the margin of the glenoid fossa. Attached only by soft tissues to the shaft is a further bony fragment consisting of the tuberosities held approximately in position by the long head of the biceps muscle. Only with difficulty can the head be recovered from its displaced position on account of the tightness of the rent in the capsule. Humeral head and tuberosities can be fixed together by a moderately thick stainless-steel wire mattress-suture passed through drill holes about three-quarters of an inch apart. An incision down to bone between

SHOULDER

the drill holes allows the wire to disappear below the surface of the articular cartilage as it is twisted tight. The injury is rare but is sometimes seen in the younger adult. In time bony union occurs. After operation the shoulder requires to be in rest for at least a month. Restoration of movement is not rapid but in time is almost complete (Fig. 40).

REFERENCES

- Badger, F. G. (1954). *J. Bone Jt. Surg.* 36B, 147.
— (1956) *Ibid.*, 38B, 771.
Böhler, L. (1937). *Die Technik Der Knochenbruchbehandlung*. Wien: Verlag von Wilhelm Maudrich.
Evans, E. M. (1949). *J. Bone Jt. Surg.*, 31B, 477.
Madsen, E. (1955). *J. Bone Jt. Surg.* 37B, 241.
Perkins, G. (1953). *J. Bone Jt. Surg.*, 35B, 521.
Thomas, J. (1949). *J. Bone Jt. Surg.*, 31B, 134.
Wilson, J. N. (1954). *J. Bone Jt. Surg.* 36B, 142.

CHAPTER 12

FRACTURES NEAR OR INVOLVING JOINTS OF THE LOWER LIMB

F G BADGER

JOINTS of the lower extremity require stability and a reasonable range of movement. Treatment of fractures involving joints aims at securing good ligaments for stability combined with the minimum of articular surface irregularity since this may predispose to arthritis.

TARSUS

Most fractures of the tarsal bones involve damage to joints and important ligaments.

Navicular

The navicular can be fractured on its own, the upper part of the bone being broken off and displaced upwards to form a bony prominence on the dorsum of the foot. Manipulation can replace the fragment but the reduction is usually unstable and open reduction with fixation by screwing or wiring gives certain union with early restoration of function.

Calcaneum

Fractures of the calcaneum mostly involve the subtaloid joint with an element of subluxation of the mid tarsal joint. Treatment was adequately described by Essex-Lopresti (1952) but some fractures with depression of the articular surface warrant more than the "propping up" with a spike that he described. The depressed fragment may comprise any part of the articular facet and require pegging to the undersurface of the talus by a metal pin driven in through the sole. In rare instances the displaced fragment can be screwed in place. After reduction, the resulting cavity in the body of the calcaneum can with advantage be filled with iliac cancellous chips. Obliteration of the cavity is better than reliance on the ossification of blood clot (Fig. 41).

The bad results of calcaneal fractures treated by any method are due to joint and soft tissue involvement. Plaster fixation, except perhaps by Essex-Lopresti's slipper, leads to stiffness whereas non-weight bearing exercises for a period of 3 months give a mobile foot, flat in appearance but often painless and serviceable for many years. The onset of progressive or continued pain and stiffness referable to the subtaloid joint may cause the patient to seek relief. Subtaloid arthrodesis can give a painless foot. Sometimes, however, pain recurs, but this time in the mid tarsal region where the joint, injured at the time of the original fracture, has broken down under the strain of taking over the function of the subtaloid joint. For this reason it may be desirable to combine mid tarsal resection with subtaloid arthrodesis. It

TARSUS

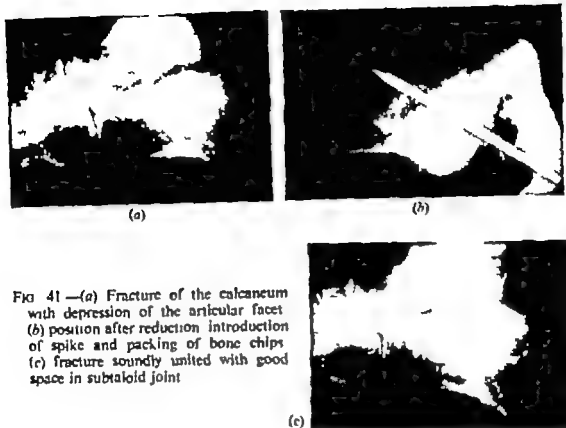


FIG 41—(a) Fracture of the calcaneum with depression of the articular facet (b) position after reduction introduction of spike and packing of bone chips (c) fracture soundly united with good space in subtalar joint

may be that reduction of a fracture can contribute to easier stabilization at a later date by improving the anatomy of the calcaneum. Selection of cases for spike reduction, open reduction, bone grafting or conservative treatment should limit the number of secondary operations.

Beak fractures of the calcaneum involving the calcaneocuboid joint, may not unite if rested in plaster. Operation may be necessary and two types of fractures are found: one where a small bony fragment needs excision and the other where the fragment is large enough to be fixed back in place.

Talus

The talus, situated between the calcaneum and the tibia, is subjected to compression between these bones and can be damaged by dorsiflexion or plantar flexion of the foot on the leg. Such injuries occur in flying and motoring accidents. Often the injury takes the form of a fracture-dislocation with a bony prominence over which skin is stretched or compressed. Despite early reduction or operation the skin may be irretrievably damaged and sloughing will develop. The dead skin may need replacement by a full-thickness pedicle flap.

Forced dorsiflexion can produce a fracture of the neck of the talus with the head of the bone displaced upwards. Manipulation may succeed in replacing the displaced fragment which can then be held in position by plaster; often replacement can only be effected by open operation. Fixation by one screw is all that is required, but plaster for a few weeks is desirable. When the dorsiflexion violence is more severe part or whole of the body of the talus is displaced backwards and upwards, probably

by coincident forced eversion of the foot. Such injuries require open reduction and again it may be possible to hold the reduction with a single screw. The displaced piece is usually robbed of its blood supply and changes suggesting avascular necrosis can be seen by radiography within a few weeks. Revascularization may occur and weight bearing is contra indicated until radiology shows union without bony sclerosis, or until the condition of the bone has become stationary.

Forcible plantar flexion results in a fracture with displacement, rarely of the head of the talus downwards, but usually of all or part of the talus forwards. Replacement requires open operation and such fixation as may be needed.

When longitudinal compression is applied to the foot in partial equinus as in alighting from a moving vehicle or falling off a step ladder the posterior process of the talus may be broken off. Pain is complained of behind the ankle, with swelling and tenderness on each side of the Achilles tendon. The fracture is not much displaced and union occurs with rest in plaster.

Gross comminution of the talus from any cause will require excision of the body or whole bone with apposition of the tibia to the calcaneum or neck of talus and calcaneum (if the head of the talus remains *in situ* and undamaged) so that bony fusion can occur. In open injuries and closed fractures with threatened penetration of the skin the operation should be carried out as an emergency procedure (Fig. 42).

ANKLE

Fractures of the malleoli are often referred to by the generic term of "Pott's fracture". The name may be embellished by "degree" or an indication of the number of malleoli involved.

The majority of such fractures result from the same type of causative violence the extent of the damage depending on the severity and duration of the force responsible for the injury. With the foot fixed to the ground and the body weight swinging so as to rotate the leg medially the ankle is subjected to the same type of violence that would occur if the foot were forcibly externally rotated on the leg. It is convenient to consider the injury as due to such a mechanism. The talus moving outward in the arc of a circle impinges on the lateral malleolus, which may fracture below the attachment of the anterior inferior tibiofibular ligament, or a tear of the ligamentous attachment without separation of the tibia from the fibula. There is then no diastasis of the inferior tibiofibular joint, and no fracture-dislocation of the ankle joint. The fracture is oblique (not to be confused with the transverse avulsion or sprain fracture near the tip of the lateral malleolus) and usually little displaced.

If the anterior inferior ligament ruptures on impact, then the interosseous ligament and the lower part of the interosseous membrane are torn. At the same time the lateral malleolus is fractured, the direction of the bony injury being a spiral with or without comminution. The site of the bony trauma is entirely above the ligament. If the violence continues the talus can move outwards as the fibula breaks and the deltoid ligament is torn or separated from the medial malleolus. Alternatively the medial malleolus may fracture transversely at its base and usually will be pulled laterally. Further external rotational violence causes the posterior inferior tibiofibular ligament, a very dense structure, to pull off the posterior inferior margin of the tibia (the "third malleolus"). While the violence is pro-

ANKLE

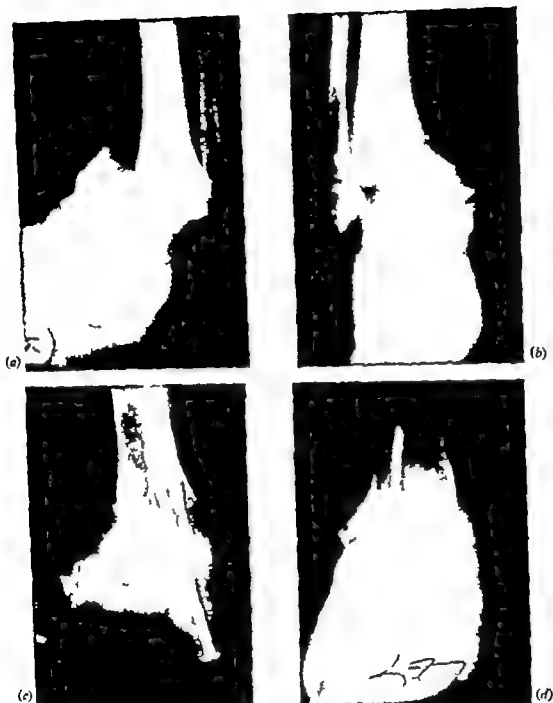


FIG. 42.—(a and b) Dislocation of the ankle with comminution of the talus (c and d) fusion of the tibia to neck of talus and calcaneum with screw inserted through sole to maintain position

during the bony damage the capsular ligament is tearing in front, so that in the fully developed trimalleolar fracture-dislocation of the ankle, deep to the skin and fascia is a concealed osteoligamentous wound involving some three-quarters of the circumference of the ankle. When the anterior inferior tibiofibular ligament has ruptured, monomalleolar, bimalleolar and trimalleolar fracture-dislocations of the

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ankle can occur from what may be considered as external rotational violence. When the interosseous extension of the deep wound is excessive and the talus comes to lie in the gap between lateral malleolus and tibia, the injury is of the type referred to as a Dupuytren fracture. If the fibular fracture is nearer mid-shaft of the bone, then the name *Maisonneuve fracture* can legitimately be used.

There are many variations on the theme" and such interesting injuries as fracture of the neck of the fibula with inferior tibiofibular diastasis or avulsion of a flake from the posterior margin of the tibia may be seen occasionally. Inferior tibiofibular diastasis without fracture of the fibula is very rare and should only be diagnosed when the whole fibula can be seen to be unfractured in the radiograph. The injury may be associated with a dislocation of the talus and usually occurs in an elderly patient.

Abduction-external-rotational injuries

Many of these injuries have been described as abduction-external rotational injuries, but true abduction injuries are uncommon and usually result from direct violence. The abduction element in external rotational injuries is incidental.

Manipulation

Manipulation can bring about reduction of most fracture-dislocations of the ankle and may result in healing of bone and ligament. Too often widening of the ankle mortice develops from recurrence of the diastasis. This happens as the plaster becomes loose with diminution of swelling. A bony redisplacement may occur necessitating one or more additional manipulations. Even when union in good position is obtained by closed reduction and a plaster cast, swelling can persist for a long time afterwards. After an interval instability may be complained of and radiological examination shows a wide mortice. In other patients the ankle may become stiff and painful after a period varying from months to years. Osteoarthritic changes can be seen in the radiograph, especially if the third malleolus was fractured and has united with a step in the articular surface and the mortice is widened.

Open operation

Open operation gives good prospects of ligamentous and bony healing in satisfactory position but frequently the operation performed fails to give the desired results. Replacement of the medial malleolus and screw fixation can give an immediate reduction of the lateral malleolus, which is pulled into position by the collateral ligaments with the talus between them. It does not necessarily restore the integrity of the ankle mortice and ligaments may heal by fibrous tissue with limited or excessive mobility. A sound anterior inferior tibiofibular ligament is the source of stability in the ankle mortice and the inferior tibiofibular joint.

The key to a stable sequel to repair is often the fibula, and the operation starts with an antero-lateral incision exposing the fibular fracture and tibiofibular diastasis. Once exposed, it can be seen that the displacement can be caused by external rotation not abduction and reduced by internal rotation. As the foot swings outwards, fracture surfaces and much of the ankle can be seen. Bony fragments, large enough to prevent complete reduction may be found between the surfaces of

the fibular fracture or lying between the posterior malleolus and the tibia when the former is involved. It can also be observed that the posterior fragment of the tibia moves with the lateral malleolus and is part of a larger "fragment" consisting of the two pieces of bone with the posterior inferior tibiofibular ligament between them. (This ligament may be seriously damaged when there is wide tibiofibular damage without separation of the third malleolus.) Reduction of the fibular fracture can be held by circlage wires. In some instances a small plate intramedullary nail bone graft or ivory peg may be needed. The ruptured anterior inferior tibiofibular ligament usually consists of two tufts which cannot be sutured but the torn ends can be made to lie in contact if a screw is inserted obliquely from fibula to tibia, transfixing the inferior tibiofibular joint lying parallel to the injured ligament and avoiding the articular surface of the tibia.

Metal staples, wire sutures and other means have been used to secure ligamentous healing. When the ligament does not rupture but avulses a piece of bone from the lateral malleolus or occasionally from the tibia fixation by wire or other suture is required.

Often the procedure described above results in replacing the medial malleolus and posterior fragment of the tibia when these coincident bony injuries have occurred. Soft tissue intervention will necessitate open reduction and screwing of the medial malleolus when palpation shows it to be sliding about and not in a stable position. Direct screwing of the third malleolus is rarely required but care has to be taken to ensure that a screw transfixing the inferior tibiofibular joint does not enter the fracture line and cause displacement when the posterior tibial fragment is large. The posterior malleolus may carry as much as one third of the distal articular surface of the tibia. After operation plaster is required, particularly when the fractured medial malleolus has not been screwed. It should be retained for long enough to allow of sound ligamentous healing—at least 6 weeks in a young person and as long as 10 weeks in the aged. Weight bearing in plaster can be permitted in the former after a month but in the latter not sooner than the seventh week.

Swelling after the removal of the plaster lasts a variable time. Removal of the transfixion screw is necessary if mobility is to be permitted at the inferior tibiofibular joint. The fibula executes a rolling movement on the tibia and so varies the shape and width of the ankle mortice to accommodate the wider diameter of the talus in dorsiflexion and the narrower diameter in plantar flexion. Although the screw can be taken out after an arbitrary period of 6 weeks removal can wait until the swelling has subsided and good mobility is restored. At that time the minor operation causes less of a setback than when performed earlier.

If plaster fixation is not used in a younger patient in whom the medial malleolus has been screwed, the limb tends to be swollen and painful for a time. Once comfortable, active movements can be started and it may be difficult to persuade the patient to use crutches for a sufficient length of time to ensure bony and ligamentous healing.

Many severe trimalleolar fractures occur in aged persons more often in women. The presence of obesity varicose veins or diabetes mellitus may raise doubts as to the advisability of operation, but there are added dangers in repeated anaesthesia for multiple manipulations when one anaesthetic for a first time repair may be sufficient. These patients need open operation more than the younger fitter persons.

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and the operation must be carried out before swelling and blistering make surgery unsafe

Abduction fractures

The true abduction fracture is seen most often in the motorcyclist, sometimes as an open injury due to direct violence producing angulation at the ankle. The lateral malleolus is fractured at joint level with comminution, but with a generally transverse fracture line. The medial malleolus is avulsed and pulled downwards and outwards. Such fractures can be treated by closed and open means depending on circumstances.

Other fractures

Forced abduction causes the talus to break off the medial malleolus by a longitudinal fracture or vertical line combined with a fracture of the lateral malleolus at ankle level. Reduction is readily carried out by manipulation, but the medial malleolus redisplaces easily and open reduction and screwing are frequently necessary.

Violent internal rotation of the foot on the leg causes a fracture of the lower end of the fibula oblique and with forward displacement and separation of a postero-medial fragment of the tibia carrying about two-thirds or more of the medial malleolus. The injury is rare and usually requires reduction under direct vision with internal fixation.

Non union of the medial malleolus can occur after a variety of ankle injuries. Treatment is best carried out by drilling across the fracture line from malleolus to tibia and pegging the malleolus in place with two dowels of bone cut from the neighbouring part of the tibia. After operation the layer of fibrous tissue between the parts of the tibia is soon replaced by bone.

Falls from heights, in addition to causing fractures of the calcaneum or talus, can produce in some patients a fracture of the lower end of the tibia with involvement of the ankle joint, both talus and calcaneum escaping injury. The fracture takes two forms. The first type splits off the anterior margin of the tibia with forward and upward displacement of the talus suggesting a combination of compression and forced equinus. If there is no marked comminution the fracture can be treated by open reduction and screwing. When comminution is marked the treatment is similar to that of the second type of fracture in which there is a generalized comminution of the lower end of the tibia with angulation and a fracture of the lower end of the fibula. This injury requires skeletal traction by a wire or pin passed through the calcaneum. With continuous traction on a Braun's frame or Thomas splint a reasonable position can be achieved. After a suitable period plaster can be applied although a second pin may be required through the tibial tuberosity.

Progress is generally disappointing with poor mobility of the ankle, pain and swelling. Most patients need arthrodesis but the operation is easier if alignment has been restored by early treatment (Fig. 43).

In the child, an external rotation fracture may occur as a fracture-separation of the lower fibular epiphysis with negligible displacement, the Tillaux fracture (corresponding to injuries to the anterior inferior tibiofibular ligament) or fracture-separation of the lower tibial epiphysis with backward displacement and an associated greenstick fracture of the lower end of the fibula with backward angulation.



FIG. 43 —(a and b) Comminuted fracture of the lower end of the tibia with dislocation of the ankle (c and d) position after nailing of the fibula and manipulation of the tibial fracture. (*Cont overleaf*)



FIG 43 cont—(e and f) Arthrodesis of the ankle for post-traumatic osteoarthritis.

This last injury can usually be reduced by manipulation under anaesthesia, the displacement being corrected by internally rotating the foot on the leg. When the injury occurs in later adolescence this manipulation may fail. If so the postero-lateral part of the heel is rested on a sandbag and a sharp push administered on the lower one third of the leg in a directly backwards direction will overcome the deformity. Union in good position will result if the leg is rested in plaster.

KNEE

Any of the four bones associated with the knee can be injured with joint involvement. In the absence of injury of the head of the tibia, a fracture of the upper end of the fibula is significant as regards the knee joint only when there is avulsion of the tip of the styloid head of the fibula. This injury revealed by radiography is an avulsion of the external lateral ligament and only occurs when there is a major rupture of the soft tissues on the lateral aspect of the knee. The leg can be abducted on the femur and the widening of the joint shown by radiography. A flake avulsed from the medial aspect of the head of the tibia can indicate a corresponding rupture of medial structures, confirmed radiologically when the leg is abducted on the thigh. Both injuries are surgical emergencies, repair being carried out while tissues are elastic and not discoloured. Structures requiring suture are cruciate ligaments, capsular ligaments, collateral ligaments and capsule. Torn menisci require excision. In rare cases the peroneal nerve may be damaged, but it is usually shredded rather than ruptured. Suture is almost always impossible. Exploration for medial and lateral tears of the soft tissues of the knee joint is carried out through appropriate bayonet

incisions. After repair of such injuries, retention in plaster for about 2 months is essential. A completely stable joint with full or very good mobility can be expected. Permanent effects result from irreparable peroneal nerve injuries and may necessitate later reconstructive operations or in older patients retentive apparatus.

Head of tibia

When a force is applied to a joint and the ligament tears, the bone may escape severe injury. This happens in young adults up to the age of 40 years. After that the ligaments being stronger than the bones osseous damage results and the ligament may remain intact. Thus when the leg is forcibly abducted on the thigh by a blow on the outer side of the knee with the foot fixed or from falling so that the foot strikes the floor while the body weight causes relative abduction of tibia on femur the resulting injury will depend on the greater strength of ligament or bone. This will manifest as rupture of the medial ligament and other soft tissues or as a fracture of the lateral plateau of the head of the tibia by impact against the harder femoral condyle, producing a depression corresponding to the shape of the femoral condyle with comminution of the tibial articular surface. In addition there may be a fracture of the neck or head of the fibula. Usually the lateral meniscus is torn from its peripheral attachments and driven into the depression in the head of the tibia.

Fractures of the lateral plateau of the head of the tibia with depression in the majority of instances can be reduced only by open operation. Böhler (1933) recommended skeletal traction combined with compression by his *redresseur*. Manual traction together with a tightly applied Esmarch bandage followed by local hammering has been advised. Parker (1939) emphasized the importance of the lateral meniscus lying in the fracture line and showed that it could only be removed by open operation after which the fracture could be reduced once the "rubber buffer" had been removed from between the two fragments. He tried fixing the displaced main fragment with bone pegs. Open reduction and screwing has been used in some instances. Danis (1949) has used nut and bolt fixation. Palmer (1951) advised a transverse incision allowing an entry below the meniscus. Watson-Jones (1943) expressed the opinion that operation was not worth while. More recently skeletal traction combined with early active movement has been advised.

Provided that the patient is in good health for his age under 70 years and with out any evidence of marked osteoarthritis, operation is worth while. The fracture is exposed by a long antero-lateral incision which opens the knee joint but the extent of the fracture cannot be seen until the torn lateral meniscus has been lifted from the fracture line and excised. The greater part of the lateral plateau can be hinged outwards on intact soft tissues situated posteriorly but an osteotome may be needed to complete the anterior fracture line. Depressed pieces of articular surface can be replaced and held in place by closing up the fracture. Fixation is best carried out by means of a moderately thick wire mattress-suture introduced by means of two cannulae. Such a suture can be used when the bone is too soft or too thin for screws or bolts. The compact bone on the outer aspect of the tibial head can be reinforced by a cortical bone graft cut from the upper tibial shaft. The suture is inserted into the cannulae through a medial stab wound. Afterwards, by cutting a small window cancellous bone chips from the ilium can be packed in below the displaced pieces of articular surface, filling up a cavity which may be as large as a hen's egg (Fig. 44 and 45).

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FIG 44—Fracture of the lateral plateau of the head of the tibia. (a) Exposure of the tibia (b) torn lateral meniscus lifted from the fracture line (c) fracture line with depression of part of the articular surface

KNEE



FIG 44 *cont*—(d) fracture hinged open revealing depressed fragment (e) fragment replaced, fracture closed and cannulae inserted (f) wire passed into cannulae through medial stab wound (*Cont overleaf*)



FIG 44 *cont*—(g) wire twisted and supplementary wire suture inserted bone grafts being packed in through a "window" (h) grafts being pushed in by mallet and punch (i) the completed repair

KNEE



(a)



(b)



(c)



(d)

FIG 45—Radiographic appearances of injury shown in Fig 44 (a and b) Before operation (c and d) 2 weeks after repair

FRACTURES NEAR OR INVOLVING JOINTS OF THE LOWER LIMB

After operation a pressure bandage and a back splint are required for about 2 weeks. Within a couple of days active quadriceps exercises can be started and flexion after a fortnight. A back splint is advised at night for about a month. Weight bearing can be permitted when healing is satisfactory usually 3 months after operation. Such treatment prevents the gross knock knee deformity and osteoarthritic changes which follow bony healing in deformity. When operation is contra indicated early active movements should be prescribed but crutches will be necessary for about 12 weeks. Such treatment gives a mobile but deformed knee with reasonable stability.

Reduction, fixation and grafting give a stable knee, little or no deformity and at least 90 degrees of active flexion from full extension.

When both plateaux of the tibial head are fractured, the injury usually takes the form of a comminuted "T" or "Y" fracture. In a few younger patients internal fixation can be achieved, but usually the bone is too comminuted and skeletal traction with or without early mobilization is the better line of treatment. In the older patient realignment of the limb and plaster immobilization give the best results.

In children there is an injury to the knee which follows jumping or falling from a height landing on the feet and pitching forwards on to hands and knees. The affected knee is swollen with a tense effusion and held partly flexed. Movements are restricted and painful and there is deep tenderness to either side of the patellar ligament. Radiography shows an avulsion fracture of a small piece of the tibial spine with a moderate gap. If the knee can be hyperextended under anaesthesia and fixed in that position in plaster the radiograph will show reduction of the fracture in most instances. Before hyperextension can be carried out the tension in the knee may require to be reduced by aspiration of the effusion with full aseptic precautions. Union usually occurs within 6 weeks and movements soon return with active use. When this manoeuvre is not successful open reduction and fixation of the bony fragment is required.

At times various pieces of the tibial head are chipped off and if large enough are amenable to open reduction and screwing. Provided that the knee is stable after operation early mobility is the best form of after treatment. Stainless-steel screws can be introduced through articular cartilage cut away over a localized area with countersinking into the underlying bone provided that the screw is not situated at a site which is regularly subjected to weight bearing.

Femoral condyles

Similarly isolated fractures of the femoral condyles can be reduced and fixed, but screws should be removed from "permanent" weight bearing areas (see Fig. 52, Chapter 13).

One or other femoral condyle may be broken off or the condyles may be split apart with distortion of the articular surfaces in "T" or "Y" fractures. Where condyles are split apart, or displaced so as to cause a step open operation and screwing is the ideal treatment, even in the older patient with early osteoarthritis. If in a "Y" or "T" fracture, after restoration of the anatomy of the lower end of the femur the condyles can be firmly attached to the shaft so that early active knee movements can be carried out, so much the better.

After operation on the lower end of the femur the Thomas splint with a hinged knee piece is the most convenient fixation allowing rest with the knee partly flexed but also permitting early movement. Straight leg raising has no virtues and throws too great a strain on a fracture of the femur. Provided that the limb hangs immobile without weight bearing crutches can be provided once union is progressing satisfactorily even if the patient spends the remainder of the day with the leg suspended on the Thomas splint.

Rarely gross comminution of both femoral condyles, or even the whole of the lower end of the femur is encountered usually as one of multiple injuries. No reduction is possible and arthrodesis of the knee by bone grafts from the femoral shaft to the tibial head gives the best limb. If the injury is open closure of the wound with local and systemic antibiotic therapy and splintage is the best emergency treatment.

Once healing is sound, arthrodesis of the knee by bone grafting can be carried out. After such grafting splintage is necessary for some months and a caliper is required for weight bearing until fusion is sufficiently sound (Fig. 46).

Patella

Fractures of the patella involve the knee joint and vary from a fissure to wide separation of fragments. The level of injury progresses upwards from an avulsion of the lower pole to tearing away of the quadriceps tendon with a few attached osteophytes. The level of the fracture tends to rise with age the avulsed osteophytes occurring in the patient of mature years.

Depending on the causative violence the fracture may be transverse with or without fragmentation of the upper or lower fragments stellate or grossly comminuted. The last two types occur as a result of direct violence and the injury may be open. Direct violence in the aged especially when osteoarthritic changes are present in the patellofemoral joint, may produce a transverse or less commonly a longitudinal fracture without much separation or obvious comminution. The longitudinal fracture may separate only the lateral or medial margin of the bone.

Treatment of patellar fractures aims at the restoration of the extensor mechanism of the knee. Transverse fractures in older patients without wide displacement, heal well in plaster. Longitudinal fractures need a supporting bandage and possibly a back splint for a few days. All of these patients can walk straight away and, with advantage practise quadriceps exercises from the beginning of treatment. Other fractures require surgery and many operations have been advised, varying from total excision, purse-string sutures for stellate fractures and the use of bolts and screws for transverse fractures. The patella is necessary for the full efficiency of the extensor muscles of the knee—its removal alters the line of pull of the muscles and reduces their effective power by at least 15 per cent. Degenerative changes in the patellar articular facet of the femur may ensue upon total or partial excision, and are more marked after total excision. On the other hand, removal of all or part of the patella converts a fracture into a soft tissue injury with consequently quicker healing. Review of a large number of patellar fractures treated at the Birmingham Accident Hospital showed that aseptic necrosis of the patella affected only the upper fragment of a transverse fracture treated by open reduction and suture. The findings suggested that longitudinal drilling for suture cut off the blood supply from

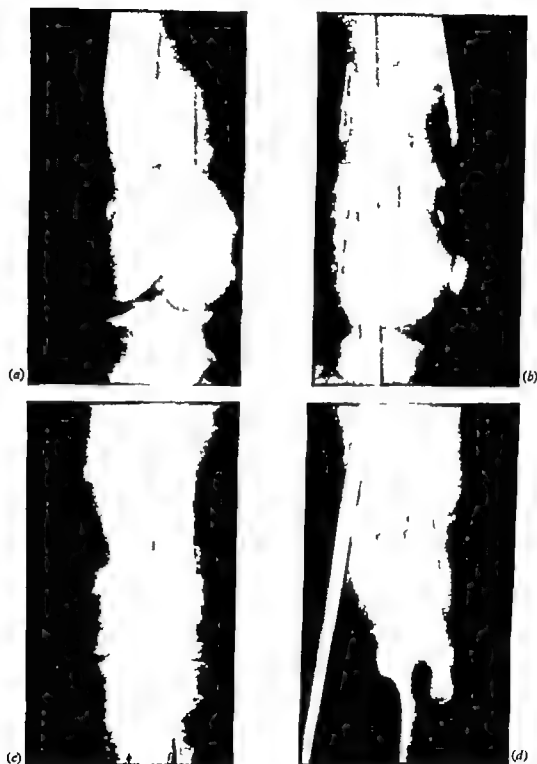


FIG 46—(a and b) Gross comminution of femoral condyles and lower part of the femoral shaft (open injury) (c and d) arthrodesis by cortical bone grafts following healing of original wound.

medial and lateral arteries. Healing of a patellar fracture tends to be slow in favourable circumstances and is much slower when aseptic necrosis has developed. Suture should be reserved for transverse fractures in younger athletic persons who can be relied upon to co-operate fully in their rehabilitation. Internal fixation should only be carried out if an exact reduction can be obtained and held until healing is complete. There is a place for screwing or fixation by nut and bolt with plaster for a short time or no plaster at all. If reduction cannot be obtained, the smaller fragment should be sacrificed, the extensor mechanism being restored by apposition of soft tissue—patellar ligament or quadriceps tendon—to raw surface of bone by sutures passed through drill holes in the bone. If the fragments are of equal size the lower fragment should be retained. Where one fragment is comminuted the remaining fragment is preserved. Even half-an-inch of patella can be used and in a female patient gives a better cosmetic result preventing the broad "knobbly" knee resulting from total patellectomy. Whatever else is done the tears of the vastus medialis and vastus lateralis require careful repair.

The author favours a transverse incision and if part or all of the patella is to be excised the heel is rested on a sandbag. While soft tissue is being apposed to bone, or patellar ligament to quadriceps tendon, pressure on the head of the tibia by an assistant hyperextends the knee a little, thereby facilitating the tying of sutures. After operation the limb in plaster is rested for 5 days, after which crutches are allowed, no weight bearing is permitted, nor active attempts at muscle contraction. Quadriceps exercises are not started before the tenth day. Following removal of sutures and replaster on the twenty-first day weight bearing is started. If plaster is retained for 6 weeks, extensor lag is never encountered. Good movements are restored by active use and exercises. Avulsion of osteophytes from the upper border of the bone is associated with a hard avascular condition of the upper part of the patella. The bone of the upper pole requires to be cut away until vascular cancellous tissue can be apposed to the avulsed end of the quadriceps tendon. This injury is not associated with tears of medial and lateral expansions, but if not repaired there is a persistent weakness of extension, the limb tending to give way.

To prevent danger to blood supply sutures can be passed through drill holes at right angles to each other in the patella, the resulting bony corner being broken down with a bonehook so that a curved needle can follow an angled track through the bone (Fig. 47).

In selected cases, suture of the patella—that is, the preservation of the whole bone and restoration of the extensor mechanism—can give a result that is virtually 100 per cent satisfactory. Suture in the unsuitable knee can give the worst result imaginable. Partial excision usually gives a knee function of about 90 per cent as measured by power of extension and range of movements. Where only the extreme lower pole is excised in a fracture not involving the articular surface, the result can be expected to be 100 per cent satisfactory as the injury tends to occur in younger patients. Total excision rarely gives a result better than 85 per cent. In the series surveyed at the Birmingham Accident Hospital, one patient after total patellectomy was rated 100 per cent. The operation was in a youth of 16 years for gross comminution. Being an ardent footballer he worked hard to regain a strong knee with full power and movements so as to resume his football. He achieved his ambition. By measurement, the circumference of the lower thigh in the affected limb was 2 inches greater than that of the normal limb, so much was the muscle power

FRACTURES NEAR OR INVOLVING JOINTS OF THE LOWER LIMB

increased in order to overcome the mechanical loss of efficiency of the extensor mechanism brought about by the removal of the patella. Total excision is sometimes required in older persons who have transverse fractures with wide separation and osteoarthritis of the patellofemoral joint

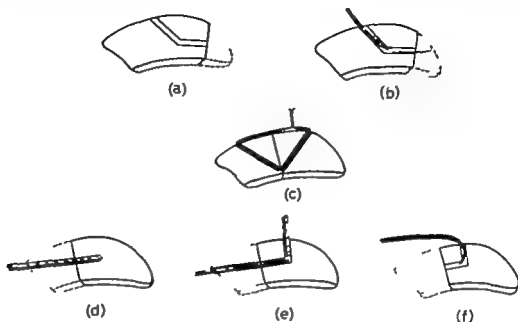


FIG. 47—(a and b) Method of drilling patella for use of a curved needle (c) method of drilling patella and insertion of wire for repair of transverse fracture (d, e and f) method of drilling patella for use of a small round needle.

HIP

Fracture-dislocations of the hip occur as dashboard injuries. The femoral head is driven backwards and in dislocating breaks off the posterior part of the acetabulum the displaced fragment may carry as much as one fifth or more of the articular surface. The dislocation can easily be reduced by manipulation but the fragment remains displaced and may be so rotated as to have a sharp edge which damages the sciatic nerve lying behind it. These displaced fragments are better replaced by open reduction and screwing. The operation should be carried out before tissues become too oedematous. If the sciatic nerve is not damaged, reduction of the dislocation can be undertaken as soon as convenient, open treatment of the fracture being carried out within the next 48 hours.

Sciatic nerve damage is an indication for early manipulation followed immediately by open reduction of the fracture and repair of the sciatic nerve if practicable. After open reduction and screwing of the fracture traction on the limb is desirable for a minimum of 6 weeks. When the fracture-dislocation is painful and the patient nervous, restless, or unable to co-operate owing to other injuries examination for sciatic-nerve damage must be postponed, but manipulative reduc-

tion of the dislocation should not be delayed. In spite of treatment permanent disability can result from irreparable nerve injury and aseptic necrosis of the femoral head can follow injury to blood vessels sustained at the time of the accident.

REFERENCES

- Böhler L. (1933) *Die Technik der Knochenbruchbehandlung*. Wien: Verlag von Wilhelm Maudrich.
 Danz, R. (1949). *Theorie Et Pratique De L'Osteosynthese*. Paris: Masson et Cie.
 Essex-Lopresti, P. (1957) *Brit J Surg* 39: 395.
 Palmer I. (1951). *J Bone Ji Surg* 33B, 160.
 Parker A. O. (1939) Annual Meeting of the British Orthopaedic Association in London.
 Watson-Jones R. (1943) *Fractures and Joint Injuries*. Edinburgh: Livingstone.

CHAPTER 13

INTERNAL FIXATION OF FRACTURES

J. H. HICKS

This chapter is mainly concerned with the prevention of joint stiffness through the internal fixation of fractures. By "fixation" of a fracture ought to be meant prevention of movement at the fracture site (that is, relative movement between one fragment and the other) at all times. In practice, to many surgeons it simply means the prevention of a visible deformity when the fracture has united. A clear distinction is necessary. Movement at the fracture site may occur repeatedly without ever producing visible deformity; it may be of an amplitude too small to be revealed by the comparatively crude techniques of clinical radiography or it may be alternating in opposite directions and not detectable when the limb is examined. Confusion between the two meanings has resulted in Watson Jones (1952) advocating "complete continuous immobilization by a method—the plaster cast—that many surgeons regard as being incapable of providing it, while Perkins (1953) attributed to "rigid immobilization" the bad result of what many would regard as a manifestly imperfect fixation. The definition of fixation must be made much more strict. Splintage used merely to prevent visible deformity should be regarded as a very low standard of fixation. This kind may merit much of the criticism levelled at the concept of fixation in general.

For convenience, angulation will be the movement usually referred to, but this may be taken to imply comparable degrees of rotation, shift and compression-distraction. External splintage is moderately good at preventing visible deformity but it cannot be guaranteed to reduce movement at the fracture site to less than 10 degrees of angulation. Conventional internal splintage cannot be guaranteed to reduce movement at the fracture site to less than 5 degrees of angulation. The cases cited in this chapter as having been "rigidly fixed" have probably never shown more than 1 degree of angulation, some of them considerably less. The results give some impression of what is being achieved. Theoretical reasons for good final function are complex; it is not due simply to freedom of joints from the immobilization of supplementary splintage. It seems to devolve from a basic principle that freedom from stiffness comes from a complete absence of movement at a fracture site.

RIGIDITY

Despite the literature devoted to the subject of joint stiffness the secret of preventing it still seems to evade the average surgeon; as indeed, to judge from some of their published results, it seems to evade the masters. The theme of this chapter is that the hitherto unsuspected cause of stiffness is low-amplitude movement at the fracture site, its presence or absence making the difference between the bad and the good results. This harmful movement may be of the order 1–5 degrees of angulation.

RIGIDITY

Absolute fixation that is elimination of all movement between the fragments is unattainable. For purposes of treatment "absolute fixation" could usefully be defined as the absence of any more movement than normally occurs in an unfractured bone as the result of its normal elasticity. This can theoretically be achieved by minimal forces or by maximal rigidity. The latter should produce this degree of fixation even in a completely unstable fracture in the presence of such forces as are unavoidable. Even this may be unattainable because bone is so strong that splints of comparable strength would be too bulky. A considerable improvement on present standards is, however, possible.

Measurement of the movements occurring in splinted fractures (Hicks, unpublished work) has shown that ordinary plates and nails do not provide much rigidity unless they receive considerable assistance from the inherent stability of the fracture. Thus the average Kuntscher nailed femur probably bends 2 degrees every time a straight leg raising exercise is done and a plated tibia may bend 5 degrees every time weight is borne unless the line of weight falls within half-an-inch of the plate. External splintage does not help. It offers almost no resistance to the first 5 degrees of angulatory movement and only slight resistance over the range of 5-10 degrees. It thus does not begin to act as a supplementation until grosser ranges are reached.

Desirability of rigidity

At present there are two schools of thought as regards the desirability of rigidity: one somewhat fatalistic and the other critical. Watson Jones accepts that not much rigidity is produced by conventional internal splintage even though a certain amount may be desirable and prefers to call it "internal suture". He teaches that external supplementation is always necessary. This is true for the majority of present day internal splints but considerable improvements could be made if the need were accepted. Charnley (1953) on the other hand doubted the wisdom of even attempting "absolute immobilization" because it might be an artificial state of affairs contributing to delay in fracture union. In support of this he drew attention to the minimal callus that appears in these cases. Union with minimal callus, however far from being undesirable, is the most efficient form of union as regards final function and is certainly no slower than union with much peripheral callus. That this is not recognized is probably due to the rarity with which fixation is done rigidly and the difficulty of differentiating the radiological appearances of union with minimal callus from one particularly vicious form of non union (details of which cannot be entered into here). On the more philosophical plane Charnley is on questionable ground in implying that rigidity is unnatural. Far from being unnatural, the normal state in an unfractured bone is a rigid continuity between one portion and another and it is towards the re-establishment of this state that all natural healing processes seem to be directed.

Influence of corrosion on design of plates

Several workers have acknowledged that conventional methods of internal splintage are not always rigid and have introduced improvements. Slowness in developing strong designs has probably been due to fear of metal and is closely linked with the history of corrosion. The original Lane plate was weak and subject to breakage (Sherman, 1912) but it was later modified and given a much stronger

cross-section, being then one of the most rigid splints that has ever been in use. It was however made in corrodible steel and it was probably the results of this that eventually brought the whole method into disrepute. The early stainless steels were little better and during the 1930s it became the fashion to use the smallest possible appliance if internal fixation had to be done at all. That this was a forlorn hope is evident when it is realized that only a few milligrams of corrosion may be sufficient to lead to wound breakdown. All that was achieved was highly inefficient splintage using implants that presented a relatively larger surface area to the surrounding corrosive fluid and nevertheless contained enough metal to produce a corrosion reaction many times over. The Sherman plate has a similar history (Sherman, 1912 *Commercial Standard* 37-31 1931) but it survived through the stainless-steel period and was further strengthened because of early breakages when Vitallium was introduced. This alteration was received without comment by the profession and the fact that the increase in the bulk of metal was not followed by an increase in wound breakdown has probably been obscured by the continued use of old and corrodible metal whilst stocks lasted—and stocks have lasted in some cases until the present day. Metallurgical requirements have been fully discussed elsewhere (Clarke and Hickman 1953). If proper precautions are taken to ensure that modern metal is used almost complete freedom from corrosion can now be ensured. The need to limit the quantity of metal is therefore no longer an excuse for disregarding good engineering practice.

Methods of increasing rigidity

From the engineering point of view the possible methods of increasing rigidity are (1) the use of two plates—one on each side of the bone (2) to revise the design and increase the dimensions, particularly the thickness of the plate and (3) to use a more rigid alloy.

Two plates

Wenger (1946) Harrison and his colleagues (1949) and Peterson and Reeder (1950) were amongst those dissatisfied with the rigidity of conventional plating and they advocated two plates. The present writer's measurements show that this common sense method is indeed the most efficient—the rigidity produced by two particular Sherman plates was 16 times as great as that produced by either plate alone—but it suffers from the disadvantage of an extensive surgical approach.

Increase in size of single plate

The use of a single plate of increased size is limited by considerations of bulk and the need to close the wound over the appliance. T and H sections are therefore excluded. Venable and Stuck (1947) Peterson (1947) and Burns have all produced designs intended to be stronger than the corresponding Sherman plates but the measured increases are not great and in some cases are more than swallowed up in the variations resulting from lack of standardization in thickness in manufacture. The only plates that possess any considerable degree of rigidity are seldom used. One is Stamm's diamond shaped plate which suffers from being rather short for bridging a comminuted segment. Its great width is also a possible disadvantage in that it occludes a considerable area of bone from contact with surrounding tissue.

In another design, by Danis (1949) rigidity is achieved by considerable thickness strong screws and engineering precision of manufacture His plates also incorporate a means of causing compression which, irrespective of whether it directly influences fracture behaviour can materially increase the rigidity produced Criticisms of the Danis plate are its shortness so that unduly heavy strains are imposed on the screws, and that the screws are so large (about $\frac{1}{4}$ inch diameter) that they may constitute a danger to the viability of the smaller bones

Design of the plate

Experimental work has shown that the movement between the fragments which occurs when a plated fracture is put under stress is a composite of (1) the springiness of the central shank of the plate (2) the springiness or the bend of the narrow strips of metal on either side of the screw hole at either end of shank and (3) the give in the hold of the plate upon the bone itself a composite result of bending of the necks of the screws and play of the screws in the plate holes Of these (1) can be reduced by increasing the thickness of the plate and (2) can be eliminated by excluding holes from the main structure of the plate

The remedy for (3) is intimately associated with another factor namely the control of rotatory movement at the fracture site Hitherto the discussion has been confined to angulatory movement because this is the type that most often occurs but rotatory movement is next in importance If angulation and rotation are satisfactorily dealt with the other types of movement (shift and compression distraction) will always be under control The straight line arrangement of the screw holes in conventional plates is inefficient in controlling rotation because it does not prevent rocking of the plate on the bone Staggered screw holes the screws entering at different points in the circumference of the bone, are probably the best answer This feature is only found in Stamm's plate in a plate designed by Durao (1952) and in a rather weak zig zag plate and a heavy-duty Venable plate that have recently appeared in the catalogues

The offsetting of screwholes in lugs on either side of the plate therefore provides an answer to both (2) and (3) and these features are incorporated, together with increased thickness and certain points dictated by biological considerations (see below) in plates for the radius and tibia designed by the author The radius plate is more rigid than any plate ordinarily used. It has now been used in a trial series of 50 cases over a period of 3 years and results have proved to be satisfactory The lug plate for the tibia is over 3 times as rigid as any plate ordinarily used. Results are promising but it is still too early to recommend it for general use

In discussing the design of very rigid plates it is not intended to imply that their use will be necessary in every case or even in the majority of cases. Considerable rigidity can be produced by ordinary plates if there is some inherent stability in the fracture The requirements are that the fracture shall be reduced to a "hair-line" fit, that little or no comminution shall exist, that there shall be an intact soft-tissue hinge on one side of the fracture and that the plate shall hold the opposite side tightly closed (Fig. 49) A crude test at operation is to rough handle the limb without any visible "give" occurring at the fracture line When the soft tissues are too much damaged to form a hinge or if comminution or missing pieces prevent a firm butting together of the fracture ends or if perfect reduction is not achieved an

INTERNAL FIXATION OF FRACTURES

ordinary plate will give only a non rigid fixation and it is in these circumstances that a really rigid one is required. In our experience this occurs in about 1 in 5 cases but the proportion may be smaller in other clinics where smaller numbers of severe fractures may be encountered.

BIOLOGICAL SIDE EFFECTS

There are some reasons to suppose that death of bone is a harmful influence in the healing of fractures and that stripping of periosteum may contribute towards death of bone. It could even be inferred from a recent article (Cohen and Harris, 1958) that the blood supply entering via the periosteum might be essential to the life of the cortex. If this is true, more emphasis ought to be placed on the periosteal source of blood and less on the nutrient artery. The original injury does a variable amount of damage to the blood supply and this is outside the control of the surgeon but if he then makes a longitudinal incision and reflects periosteum in order to apply a plate he will be adding a large triangular area of denudation which could have been avoided. Urist, Mazet and McLean (1954) placed so much emphasis on this additional operative damage that they condemned internal fixation for all badly comminuted fractures. Charnley (1957) condemned internal fixation of double fractures of the tibia because of the danger of converting the whole of the central fragment into a dead sequestrum. Neither seems to have considered that this damage could possibly be avoided or at least minimized. Our experience is happier than that of Urist (see Fig. 50) or of Charnley and it may be that we are here witnessing the better results of the non-strippers of periosteum compared with the strippers."

Although the above considerations are mostly theoretical and are disregarded by many surgeons, the author believes that they cannot be ignored and that the safer policy until such time as it is proved unnecessary is to plate over the periosteum. This is a counsel of perfection and makes the operation longer and more difficult. Where a bone is giving direct origin to muscle it is difficult even to find a layer of periosteum not to strip. In the tibia however it can always be achieved. The incision need not go right down to bone such dissection as is essential can be done superficially to periosteum the most accessible surface may be used, and the plate can be laid on top of the periosteum. In the few instances where intact periosteum hides the fracture line from view and would prevent reduction, it can be cut transversely (along the line of the fracture) instead of making a longitudinal slit and peeling the bone like a banana. The larger the plate the more important becomes this avoidance of periosteal stripping for its insertion. If one thinks in terms of damage to cortical blood supply the raising of periosteum followed by stitching it back into place is seen to be folly. With practice it becomes possible to complete an internal fixation with little or no addition to the existing periosteal stripping but the operation requires time and deliberation. It might even be undesirable to squash periosteum as occurs when an ordinary plate is screwed down tightly. A number of cases have been tried with washers between plate and bone but the technique is too difficult to be recommended. In view of this the lug plate for the tibia has the equivalent of "built in washers" making the main shank stand clear of the surface of the bone by a fraction of an inch.

This risk of damage to the blood supply is also the main argument against double

plating. The double approach or alternatively the wide retraction required to apply plates to two different aspects of a bone must do considerable damage and has probably provided the bad results that the conservative school loves to publicize. The one occasion when double plating is justifiable is when the periosteum and soft tissues are found so widely stripped by the trauma that the plates can be applied without any further dissection whatsoever. Ordinarily a plate cannot be applied to the lateral surface of tibia for example, without dissecting away muscle. The severe fracture which is most in need of rigidity is most widely stripped of muscle and can be double plated with least harm. Most gratifying results can be obtained.

With the periosteum thus cared for the only biological damage that remains comes from the screws. This is unavoidable and all that can be done is to keep it to a minimum. It is perhaps not realized that the diameter of the standard size screw is one-sixth of the diameter of the shaft of a female radius. In addition for one half to one cm. around each screw there may be a cylinder of dead bone killed perhaps by the heat of drilling. Damage to blood vessels within the bone is thus a great risk and the author has seen two cases of fractured radius fixed by standard-size screws in which a half inch length of shaft between the fracture end and the first screw hole died. Watson Jones' advice against high-speed mechanical drilling is therefore wise and in an additional attempt to minimize this hazard the radius lag plates have been arranged for use with smaller than normal screws.

RESULTS OF RIGID FIXATION

Good final function

In the author's opinion the most important effect of rigid fixation is that it minimizes joint stiffness. This is illustrated in Fig. 48 which shows an example taken from the group of fractures that can be rigidly fixed by ordinary plates. The 76-per-cent range of ankle movement shown would probably have been greater had not the wound of the navicular and ankle joint become heavily septic. This case is not selected from amongst the best results as it is intended to be representative. Other cases are shown in Figs. 49-52 and 56.

Ankle range¹ (which is representative of function of the limb as a whole) has been measured in nearly 100 cases with fractured tibias. Cases with over 80 per cent of normal range were found to be almost indistinguishable, subjectively and objectively from normal. The results from different methods of treatment are given in the Table and show that increasing rigidity produces increasing numbers of almost perfect results. The effect is even greater than would appear from these figures because of the particular criteria for assignment into the groups. These were in general, that the milder fractures were given conservative treatment and the more severe ones operative treatment, the very worst being selected for the specially rigid fixation. This is revealed in the increasing proportion of compound fractures in the three successive groups.

¹ Throughout this chapter where ankle range is quoted it has been measured radiographically and the percentage given is of the opposite normal ankle. Deficiency in knee flexion is calculated from the heel-to-buttock distances of the two legs. Inversion-eversion is assessed by clinical comparison with the opposite normal foot.

INTERNAL FIXATION OF FRACTURES

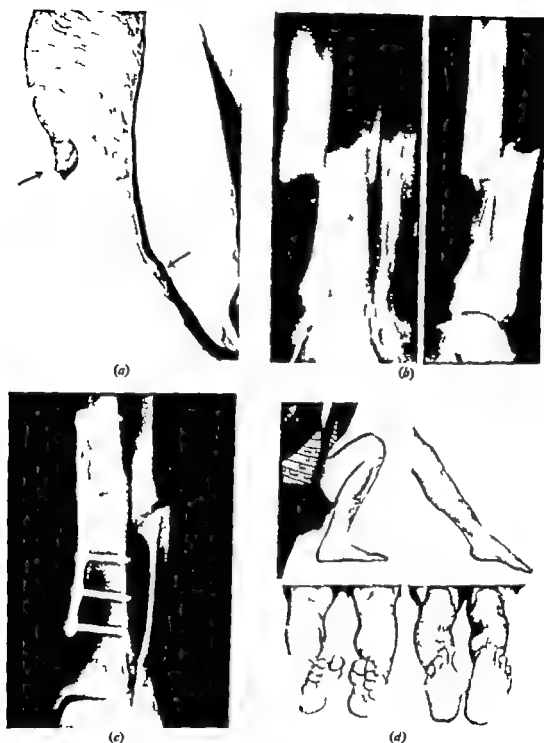


FIG. 48—A typical case treated by rigid fixation (a) and (b) Clinical and radiographic appearances of a compound "motor-cycle tibia." Two inches of bone protrude at the upper arrow and the lower arrow indicates a compound fracture of the navicular and a penetrating wound of the ankle joint (c) Radiograph at 1 year (d) Function at 1 year—knee range full ankle range 42 degrees (76 per cent of normal) inversion-eversion full the patient could run and play football

RESULTS OF RIGID FIXATION

The author does not subscribe to the idea that this freedom from stiffness is simply due to the joints being free from the encumbrance of external splintage. He believes that movement at a fracture site may have a direct effect in causing joint stiffness and that prevention of such movement prevents the inflammation and the stiffness. The rigidity however must be successful in preventing even the last few degrees of movement otherwise this effect is not seen. The joint stiffness caused by plaster the author believes is because external splintage not only permits but actually causes movement at the fracture site.

TABLE
PROPORTION OF CASES OF FRACTURED TIBIA WITH 80 PER CENT
ANKLE RANGE OR OVER AT 1 YEAR

<i>Treatment group</i>	<i>Percentage of cases resulting in 80 per cent range or over</i>	<i>Percentage of compound fractures in each group</i>
Conservative treatment	35	30
Ordinary internal fixation	60	40
Specialty rigid internal fixation	75	70



FIG. 49—Union with minimal callus. (a) Fracture capable of rigid fixation with an ordinary plate. (b) Radiographic appearance at 1 year. Ankle range was 33 degrees (77 per cent of normal), knee range minus 15 degrees of flexion (this limitation was partly or wholly due to a fracture of the femur sustained at the same time) inversion-eversion about 75 per cent of normal.

INTERNAL FIXATION OF FRACTURES

Pattern of union

The radiological pattern of healing seen when complete absence of movement at the fracture site exists is something so unfamiliar that it may go unrecognized. Callus is formed only in the minimal quantity required to fill the fracture line. External callus is noticeably absent. The fracture line simply fades away and the normal bone structure is restored as shown in Fig. 49. To Danis (1949) of Brussels must go the credit for the first clear description of this phenomenon. Freely translated he stated: "After a really rigid internal fixation (and provided it heals aseptically) callus develops in such small amounts as to be invisible. There is no periosteal callus, no intramedullary proliferation, and the fragments fuse together without any intervening tissue." This work of repair takes place without any apparent participation of the surrounding tissues. The periosteum reacts only occasionally and then only if it has been left stripped. It forms thin outlying strands only resembling cigarette smoke which take very little if any part in the real consolidation of the fracture.

Another example is shown in Fig. 50. The comminution in this case made it one of the fractures that require a specially strong plate before rigidity could be produced. It is easy to understand how anyone who requires a large external callus before he can pronounce a fracture united will be in a quandary and may think that such a case is taking a long time to unite. As Danis said, "standard teachings on union of fractures are the outcome of experience by surgeons who have only used conservative methods of treatment. It was thus that the idea arose that the production of much callus was an essential process in the union of a fracture." The author would add that it is also often the experience of surgeons who use non rigid methods of internal fixation.

Another feature in the pattern of union is the relative absence of "decalcification." A patchy decalcification, most readily noticed in the cancellous bone areas, is a bad sign, being invariably associated with joint stiffness. It is usually a well marked feature of conservative treatment and to a lesser extent of non rigid internal fixation. It is less often present and is less severe with rigid fixation as can be seen in the examples cited.

THE INTERNAL FIXATION OF COMPOUND FRACTURES

The unstable compound fracture is not dealt with satisfactorily in the standard teachings, for while compounding is taken to necessitate a reversion to conservative treatment, the accompanying severe instability is left inadequately dealt with. The conventional argument is that although stabilization may be desirable the danger of foreign bodies in contaminated wounds provides an overriding objection. The result in many cases is a low level of fixation and in consequence a poor final function.

Venable and Stuck in 1941 claimed that internal fixation for compound fractures was fairly safe if Vitalium was used, but even in 1953 Zadik's paper brought only slight concessions from leading authorities towards accepting such a line of treatment. If however only the mildest and least contaminated fractures are suitable for internal fixation, as these authorities lay down, no advantage will be seen since these are the cases that give satisfactory results by conservative methods. The fractures most in need of effective treatment—because most likely to show poor

COMPOUND FRACTURES



FIG. 50—Another example of union with minimal callus. (a) Treble compound, comminuted, "motor-cycle tibia" requiring a special plate to produce rigid fixation. (b) Radiograph at 1 year (c and d) Appearance at 1 year. Ankle range was 49 degrees (100 per cent of normal) knee range full inversion-eversion full. Note satisfactory healing of compounding wounds.

results—are the grossly compounded ones but the internal fixation of these is still generally condemned.

That the situation has changed and "sepsis" is no longer likely to be caused by surgical implants has been explained elsewhere (Hicks, 1958). Analysis of real sepsis in compound fractures has shown that the incidence is affected so little by internal fixation that the hazard of infection is no longer an overriding objection (Hicks, 1957). The teaching that a foreign body always promotes infection and that healing is impossible until it has been extruded must now be revised. It remains true of

INTERNAL FIXATION OF FRACTURES

certain foreign bodies, namely organic materials subject to bacterial attack or soluble or corrodible inorganic materials if toxic. It is not true of non irritant foreign bodies and thus holds good for surgical implants now that they are all manufactured of almost non-corrodible metal. In spite of previous warnings (Cater and Hicks 1956 Annotation 1956) some hospitals are still in possession of corrodible metalware and some surgeons therefore will still be experiencing an unnecessary amount of pseudosepsis and secondary real sepsis. When non-corrodible metal is used we would support McLoughlin (1956) who went so far as to say that internal fixation can act as a protection against infection by the prevention of small movements at the fracture line. The author is not even convinced that antibiotics account for present successes. Their introduction happens to have coincided with the gradual transition from the older corrodible metal to new and almost non-corrodible metal and thus may be the factor responsible for the improvement. Soft tissue infection can be present directly over a fracture yet will almost never spread into it and this holds good as long as the fixation remains effective. The surest way of causing a catastrophic spread is to remove the implant and allow the fracture to collapse. The non-rigid internal fixation gets the worst of both worlds. Mechanical irritation from a little looseness causes screws to work loose. This appears to afford ingress to infection that has hitherto been lying harmlessly on the surface of the plate. Then the screws work completely loose and the whole fracture breaks down.

The above refers to primary wound infection. Wound infection secondary to skin necrosis referred to in a previous publication cannot be so summarily disposed of. It is however more amenable to treatment (by skin grafting) when it does arise.

Statistics are to be found in the above mentioned publications. In Fig. 51 is illustrated a severe case from our series of plated compound fractures. The lower end of the upper fragment of this tibia was protruding through a wound on the medial side of the ankle. No photograph is available but the initial radiograph gives some impression of the bony damage. The patient a woman aged 55 years had crawled up her garden path to find help and in consequence mud and grass had to be removed from the bone. Internal splintage was nevertheless used. The wound was nearly healed at 4 weeks and soundly healed within 6 weeks. Penicillin had been given for the first 4 days although it was probably not necessary.

Other examples of the satisfactory use of internal fixation for a compound fracture are seen in Figs. 48 and 50. Healing was complete at 5 weeks in both these cases.

It must be emphasized that if the internal splintage is not rigid results like this may not be obtained. The compromise of using a minimum quantity of metal achieves the worst of both worlds.

It is evident (Hicks 1957) that all or nearly all, clinical wound infection in plated compound fractures is due to the compounding and not to the plating. One remaining question is still to be answered before a surgeon will feel free to plate compound fractures namely what to do when cases of sepsis do arise. The author is convinced that the wrong action is to remove the metal. To give reasons for this would involve too long a discussion but in practice union can and often does proceed and infection does not spread when the fixation is preserved, whereas complete breakdown of the fracture and catastrophic spread of the infection may occur if it is removed.

METAL NEAR JOINTS

Conservative treatment could scarcely have given better wound healing in the cases cited above and the functional result might well have been poorer. The present situation is that internal fixation is responsible for a severe complication in a very small proportion of cases but in exchange gives a much better function to a substantial majority. The only qualifications are that rigidity and modern metal must be used.



FIG. 51.—Internal fixation for a severely contaminated compound fracture. (a) Initial radiograph. The soft tissue lesion has been indicated. (b) State of healing and range of movement at 10 weeks. (c) Radiograph at 5 months. Final function: ankle range 53 degrees (78 per cent of normal); inversion-eversion about 75 per cent.

METAL NEAR JOINTS

The old teaching that metal near a joint causes stiffness may have been true when it set up an inflammation owing to corrosion. Inflammation from any cause near a joint results in stiffness. With modern almost non-corrodible metal this is no longer a source of trouble but the possibilities now opened up have not yet been fully recognized. One example in which metal lay within half an inch of a joint is to be seen in Fig. 51. In this case the range of movement was not greatly limited. Furthermore it must be remembered that the reason for metal near a joint is a fracture near

INTERNAL FIXATION OF FRACTURES

a joint and such stiffness as does exist might well be due to this. An example of metal actually in a joint is seen in Fig. 52. That metal driven through an articular surface may be harmless can be inferred from Bates, Reimers and Horn (1948) who described it as becoming covered with fibrocartilage. Once again, however, it must be emphasized that when metal is used in treatment of a fracture a good result probably only occurs when the fixation is rigid.

A particular difficulty with fractures near joints is that they are fractures of cancellous bone. It will be noted that whereas our technical resources for fixing cortical bone can be made quite efficient, those for cancellous bone are less good.

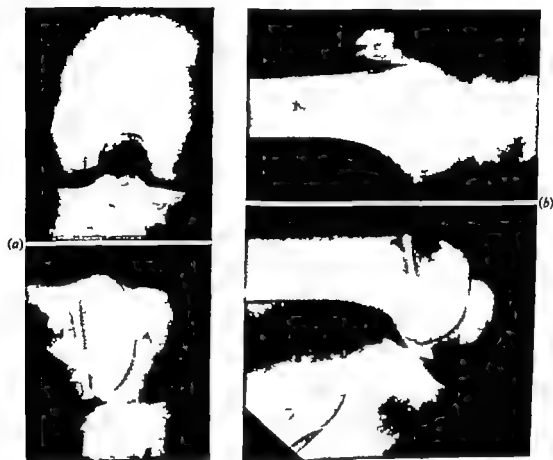


FIG. 52.—Showing that metal near a joint does not cause stiffness. (a) A screw through the articular surface to fix a fractured femoral condyle (knee semi-flexed) (b) Final range of knee movement 135 degrees (96 per cent of normal) (Mr F. G. Badger's case.)

With cancellous bone screws do not hold tightly, plates are often the wrong shape, nails work loose, and invisible cracks open up and render the fixation useless. This is not an acknowledgement of any fundamental biological difference between cancellous and cortical bone but only of a mechanical one. Thus although rigid fixation could be particularly beneficial in these sites (since there is such a large amount of stiffness to be prevented) it very often cannot be carried out, or worse, is attempted and fails to achieve rigidity.

These points apply particularly to the T or Y shaped condylar fracture of

METAL NEAR JOINTS

humerus and are illustrated in Figs. 53-56. These examples demonstrate once again the great benefit that can be achieved by fixation if it is rigid and the mediocre results from no fixation or from non-rigid fixation. They also demonstrate that only a small proportion of cancellous bone fractures are as yet capable of rigid fixation.

It is perhaps a little unfair to compare the case in Fig. 53 with that in Fig. 56 because there is no doubt that the fracture shown in Fig. 53 is the more severe fracture



FIG. 53—Condylar fracture of the humerus. A case treated conservatively. Elbow movement 52 degrees (approximately 36 per cent) (Adapted from Perkins (1953) by courtesy of the author and *Journal of Bone and Joint Surgery* and Perkins (1958) by courtesy of the author and publisher.)

but some criticism is merited because of the complacency that was shown in describing the final range of movement—36 per cent of normal—as not nearly full but sufficient for most purposes. It is ironical that the surgeon concerned categorically condemned internal fixation on the grounds that it will produce a stiff joint! It is agreed that nothing better could have been done for this patient but where it can be done rigidly fixation offers considerable advantages. Cases to

select are those in which rigidity can be guaranteed, Fig. 53 showing what can then be achieved. The penalty of misjudgment is shown in Fig. 55 and it is "failed fixation" such as this that has brought open operation into disrepute although, it may be noted this result is no worse than those achieved by conservative treatment.

The lesson conveyed by the foregoing is that internal splintage must provide absolute fixation or it is nearly always useless. With the exception noted below no compromise is any good. Half and half measures run the risks without providing the advantages. These advantages are (1) freedom from joint stiffness—which has been made the theme of this chapter—and (2) certainty of union. The latter has



FIG. 54—Condylar fracture of the humerus. A case treated conservatively. Elbow movement 50 degrees (approximately 36 per cent). (Reproduced from Charnley (1957) by courtesy of the author and publisher.)

been reserved for another occasion but it is perhaps opportune to mention that simultaneously with the results quoted herein the incidence of bone grafting for the fractured tibia has been reduced to 4 per cent. Rigidity can therefore help to solve the problem of delayed union at the same time as it is reducing the severity of joint stiffness. It should be mentioned that these results are believed to be obtained partly because of a deliberate policy of *no physiotherapy* and *late return to function* both of which factors are conducive to the prevention of movement at the fracture site. Only in those few types of fractures where stiffness and delayed union are not serious hazards and where mal position is, does non rigid internal splintage ever have a place. When rigidity is not necessary (as in the mildest

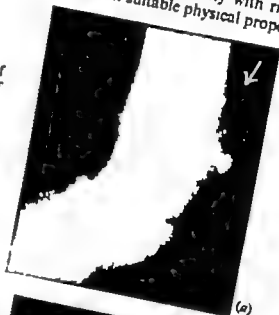
group of fractures) or cannot be provided (as in the severest group of fractures) then the next best regime is one of extreme conservatism

FUTURE DEVELOPMENTS

The advantages of rigidity are not yet widely appreciated. In fact the need for it is still contested the opposing points of view being that a little movement favours callus formation and that a fracture must be allowed when necessary to collapse into stable position. Any of these requirements namely tenacity with rigidity or with elasticity or ductility can be satisfied by metal if suitable physical properties are

FUTURE DEVELOPMENTS

FIG. 55—Condylar fracture of the humerus. A case of failed internal fixation. The arrow indicates the screw that slipped. Elbow movement 35 degrees (24 per cent), increasing finally to 65 degrees (45 per cent).



selected and appropriate modifications in design are made. Surgeons must, however first agree as to which of the requirements is the most important. Present appliances are often an unsatisfactory compromise. Agreement is also necessary on the anatomical and biological rules that must be conformed to. Despite the limitations implied and despite the very strict requirements concerning freedom from corrosion¹ certain developments are possible.

¹ These are discussed fully in *Modern Trends in Surgical Materials* Ed by L. Gillix. London Butterworth.

INTERNAL FIXATION OF FRACTURES

As regards choice of metal, if rigidity is considered of paramount importance it is doubtful whether any substantial improvement will be made on stainless steel and Vitallium. The only reason for not being quite satisfied with these is the small incidence of trouble with wound healing (about 10 per cent with stainless steel and 4 per cent with Vitallium). Further details properly belong to a discussion on corrosion. To judge from the work of Clarke and Hickman (1953) titanium should prove to be an improvement in this regard and it is already being used. Its physical

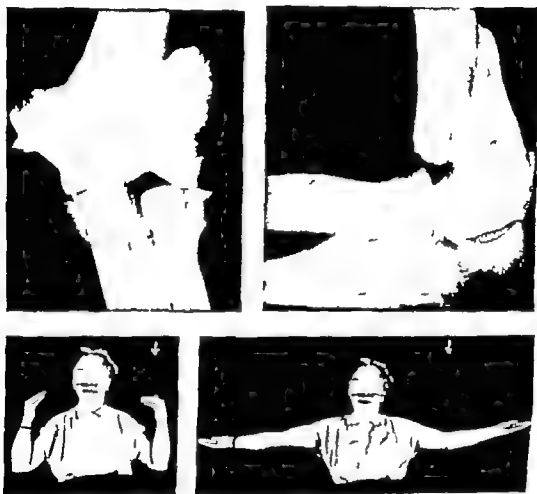


FIG 56.—Condylar fracture of the humerus. A case treated by rigid internal fixation. Elbow movement 115 degrees (79 per cent)

properties, however result in appliances being more 'springy'. Thus there is somewhat less rigidity in a plate made of titanium than an identical one made in stainless steel or Vitallium or put in another way to ensure the same rigidity a titanium plate must be made somewhat thicker than a stainless steel or Vitallium one. A few surgically untried alloys at the top of Clarke and Hickman's Table III might prove to have suitable coefficients of elasticity and since their resistance to corrosion would be satisfactory could possibly be exploited in the future. As regards the design of appliances, considerably more opportunities exist for improvement.

FUTURE DEVELOPMENTS

Although rare skin necrosis is still the most dangerous complication of fractures of the tibia and is the only factor that might force a retraction from the policy of internal fixation. It is the commonest part-cause for late amputation and there seems little doubt that its incidence is raised by the surgical incision. All other bad effects of the operation for internal splintage (such as those that follow the stripping of periosteum) can be avoided but there is no conceivable alternative to making a skin incision. External skeletal fixation if the advantages of open reduction are sacrificed can avoid this hazard. It is possible that this might provide an answer in some cases. The danger from drill holes may also be minimized since they can be placed further away from the fracture zone. Existing methods of external skeletal fixation however suffer from insufficient rigidity which technically could be improved. The future partly depends on our ability to do this without rendering the technique impossibly complicated.

REFERENCES

- Annotation (1956). "Surgical Stocks" *Lancet* 2, 879
 Bates, J. I. Reiners, C. R., and Horn, R. C. (1948). *Surg. Gynec. Obstet.*, 87, 213
 Cater, W. H., and Hicks, J. H. (1956). *Lancet* 2, 871
 Charnley, J. (1953). *Compression Arthrodesis* Edinburgh Livingstone
 — (1957) *The Closed Treatment of Common Fractures* Edinburgh Livingstone
 Clarke, E. G. C., and Hickman, J. (1953) *J. Bone Jt. Surg.*, 35B, 467
 Cohen, J., and Harris, W. H. (1958) *J. Bone Jt. Surg.*, 40A, 419
 Commercial Standard 37-31 (1931) United States Bureau of Standards.
 Davis, R. (1949). *Théorie et Pratique de l'ostéosynthèse* Paris Masson et Cie.
 Durao A. (1952) *Gaz. méd. portug.*, 5, 429
 Harrison L. M., Sawrie R. G., Neer C. S. and Craig F. S. (1949) *J. Bone Jt. Surg.* 31A, 94
 Hicks, J. H. (1957) *Proc. R. Soc. Med.* 50, 842.
 — (1958). "Pathological Effects from Surgical Metal" In *Modern Trends in Surgical Materials* Ed. by L. Gillis. London Butterworth.
 McLoughlin H. L. (1956). *Surgery* 39, 892.
 Perkins, G. (1953) *J. Bone Jt. Surg.*, 35B, 521
 — (1958). *Fractures and Dislocations* London Athlone Press.
 Peterson L. T. (1947) *J. Bone Jt. Surg.*, 29, 335
 — and Reeder O. S. (1950) *Ibid.* 32A, 532.
 Sherman, W. O. (1912) *Surg. Gynec. Obstet.*, 14, 629
 Urist, M. R., Mazet R., and McLean F. C. (1954) *J. Bone Jt. Surg.*, 36A, 931
 Venable, C. S. and Stuck W. O. (1941) *Amer. J. Surg.*, 51, 757
 — (1947). *The Internal Fixation of Fractures* Oxford Blackwell.
 Watson Jones, R. (1952). *Fractures and Joint Injuries* 4th ed. Edinburgh Livingstone.
 Wenger H. L. (1946). *Surgery* 20, 541
 Zadik, F. R. (1953) *J. Bone Jt. Surg.*, 35B, 146.

CHAPTER 14

FAT EMBOLISM

S SEVITT

A *SIGNIFICANT* degree of histological fat embolism rarely occurs except as a complication of injury. The minor degrees of pulmonary embolism which are not infrequent in diabetics and after burns and absorption of certain poisons need not be considered here.

The first accounts of fat embolism are those of Zenker (1862) who discovered fat globules in the lungs of a man with a thoraco-abdominal crush injury and of Wagner (1862) who reported 48 cases. Many cases were described during the following years but clinically the condition must often have been confused with other effects of injury. Various workers (Holm 1876, Scriba, 1880, Meeh 1892, Warthin 1913) found pulmonary fat embolism very frequent after fractures, but in many reports (among them those of Payr 1900, Gröndahl 1911, Gauss, 1916 and 1924, Vance 1931 and 1934 and more recently Robb-Smith 1941 and Warren 1946) histologically gross pulmonary embolism was equated with a symptom producing process and it was postulated that emboli in the lung could cause cardiorespiratory distress and even death. The importance of cerebral fat embolism was raised by Scriba (1880) and stressed by Warthin (1913) who claimed that a small amount of fat entering the brain might cause a severe or fatal disturbance. Many other reports, among them those of Godlee and Williams (1911), Gauss (1916, 1924), Vance (1931, 1934) and Scuderi (1934, 1938) established cerebral embolism as a clinically important entity.

The relative significance of the pulmonary and systemic forms has not been the only controversy; other discussions have centred around the origin and nature of the fat globules, their mode of action, the frequency of embolism, its diagnosis during life, and attempts to develop specific treatment.

ORIGIN AND NATURE OF FAT EMBOLI

Most workers agree that the fat originates at the site of trauma, particularly the injured marrow of fractured bones. Globules from injured fat cells gain entrance to the venous stream and the process which is influenced by orthopaedic procedures may continue perhaps intermittently for several days. Gauss (1924) suggested that embolism was likely to occur after fractures because the venules of injured marrow are held open by attachments to bone. Subsequent observations confirm that the emboli come from the sites of injury. Histology shows that most of the emboli lying globular and undistorted within small pulmonary arteries are 20-40 μ in diameter which corresponds to the size of fat cells and presumably of the droplets when first liberated. Fragments of bone marrow have been seen within small arteries in the lungs of patients showing fat emboli (Lubarsch 1893, Armin and Grant, 1951, Scully 1956, Emson, 1958) and although the

ORIGIN AND NATURE OF FAT EMBOLI

reported incidence is low this is probably because only architecturally intact fragments can be unequivocally recognized Peltier (1956a) confirmed the observations of Reiner (1907) and Caldwell and Huber (1917) that the dissemination of emboli from a fractured limb could be prevented by a tourniquet proximal to the fracture. Emboli were found in the organs of dogs with experimental leg fractures only when the tourniquet which was applied before inflicting the injury was released before the animal was killed. The tissue tension around recent fractures and injured areas is increased (Bellis 1942) and the mechanism of entry into veins is the increased pressure gradient between the injured area and the torn vessels (Young and Griffith 1950).

Fat can probably enter the blood after injury to soft tissues but the relationship between multiplicity of fractures and severity of pulmonary fat embolism (see page 220) indicates that the marrow is usually the major source. Much of the embolic fat after extensive subcutaneous bruising may come from marrow injured by skeletal shaking because manipulation concussion or jarring of bones without fracture have also been known to produce gross fat embolism. Abdominal trauma is occasionally followed by clinical embolism. Often fractures of ribs and other bones are present, but the author has seen clinical embolism develop in patients with a ruptured liver without bony injury. Presumably the damaged liver cells are the source of the fat globules which would have no difficulty in entering torn hepatic veins held open by the rigid consistency of the organ.

Criticism of the marrow theory came from Lehman and Moore (1927) who thought that the fat content of the fractured bone was often less than the total amount of embolic fat. They suggested that the emboli originate from the plasma through agglutination of the finely dispersed chylomicrons. The fat emulsion and the lipoprotein complexes in plasma were said to be broken down by lipase lipoproteolytic enzymes, or other metabolites released after injury the process perhaps being triggered off by a small amount of free fat released from the injured area. This idea was supported by Frazer and his colleagues (1945) who showed that *Clostridium welchii* toxin clumped the chylomicrons in blood. Re-examination of the fat content of human long bones has shown that the amounts present are greater than previously reported in both the tibia and the femur 100-200 g of fat are generally present (Peltier 1956b) so that there is sufficient fat in long bones to account for fat embolism. *In vitro* experiments have shown that the fat emulsion of plasma is very stable and in particular cannot be broken down by extracts of traumatized tissue. Chylomicron agglutination did not occur in the blood of rats subjected to muscle ischaemia nor in lipaemic serum from normal or ischaemic rats when extracts of muscle or liver or the serum of ischaemic animals was added (Whiteley 1954). The alarm reaction in rabbits produced by exposure to cold injection of ACTH (adrenocorticotrophic hormone) formaldehyde, or cortisone failed to produce fat embolism or an alteration in the plasma fat emulsion (Glas, Grekin and Musselman 1953). There is no necessity to postulate a plasma origin for fat emboli and the theory is based on slender evidence.

PULMONARY FAT EMBOLISM

Pulmonary emboli are found as compressed sausage like bodies within alveolar capillaries in globular form within arterioles and small arteries and as free globules

FAT EMBOLISM

in alveolar spaces but their distribution depends on the duration of survival or severity of embolism or on both factors (Grant and Reeve, 1951 Whiteley 1954). Severe embolism is closely related to severity of injury and the latter often determines the speed of dying so that it is difficult to dissociate short survival from severity of injury

Incidence and severity

Embolism commences soon after injury because histology shows many globules in the lungs of patients dying within minutes or hours of an accident. Pulmonary fat embolism can be of help medico-legally in helping to determine whether fractures occurred before or after death a minor degree should be regarded however as potentially normal. Its incidence cannot be assessed clinically but the lungs of those dying after injury nearly always contain fat emboli. The frequency ranges from 80 to 100 per cent in most of the series reported and only two authors report a lower incidence (Table I). Differences in frequency are probably related to a varying incidence of multiple and severe fractures and to different periods of survival

TABLE I
FREQUENCY AND SEVERITY OF PULMONARY FAT EMBOLISM ASSESSED
HISTOLOGICALLY IN PATIENTS DYING AFTER VARIOUS INJURIES

Author	Types of injury	Total patients studied	Incidence of severe pulmonary embolism	Total incidence of pulmonary embolism
Milosavlitch (1930)	Fatal car accidents	22	some	100 per cent
Vance (1931)	Coroner's cases	164	9 per cent	62 per cent
	Coroner's cases with fracture of lower extremity	59	20 per cent	66 per cent
Robb-Smith (1941)	Civilian casualties	115	34 per cent	91 per cent
Grant and Reeve (1951)	Civilian and battle casualties with limb injuries	25	44 per cent	100 per cent
	Battle casualties with abdominal injuries	30	10 per cent	80 per cent
Sheynis (1951)	Traffic accidents with bone injuries			88 per cent
Mallory (1952)	Battle casualties	51	20 per cent	67 per cent
Whiteley (1954)	Battle casualties	14	15 per cent	86 per cent
	Civilian casualties, mostly multiple fractures	19	37 per cent	89 per cent
Scully (1956)	Battle casualties	89	19 per cent	93 per cent
Birmingham Accident Hospital (Sevitt, 1957 Emson, 1958)	Civilian accidents	100	44 per cent	89 per cent

Those surviving longer than a week have a lower rate and a lesser degree of fat embolism than those dying earlier presumably because emboli have disappeared from the lungs

In the author's series 89 per cent of 100 patients dying after various injuries had pulmonary fat emboli but the incidence of gross embolism was mainly related to the severity of injury particularly the multiplicity of fractures of marrow rich bones (Sevitt, 1957 Emson 1958). Gross pulmonary fat embolism was found in

PULMONARY FAT EMBOLISM

18 per cent of 39 patients with mild bony injuries (fractured skull fractured neck of femur) in 45 per cent of 29 cases with moderate bony injuries (fracture of a long bone or two or three fractures) but was present in 80 per cent of the 32 patients with severe bony injuries (four or more fractures). The absence of emboli in some patients was related to a combination of mild bony injury and long survival time.

Evidence of the rate of disappearance of emboli from the lungs is obtained in patients with pulmonary infarcts at autopsy. For example, an infarct related to an episode of pulmonary thrombo-embolism on the seventh day was found in a patient who died 23 days after injury. Fat emboli were few in the normal lung and were many in the infarcted area. It was estimated that 90 per cent of the emboli present on the seventh day were removed during the subsequent 16 days.

Significance

Clinical observations

A slight or moderate degree of pulmonary fat embolism does not produce clinical effects but the significance of histologically severe embolism is in dispute. It is possible that blockage of the pulmonary bed by multiple emboli impedes the blood flow through the alveolar capillaries, produces tissue anoxia, interferes with the exchange of oxygen and carbon dioxide, leads to congestion of the pulmonary veins, perhaps to an increased pressure in the pulmonary artery and a fall in systemic arterial pressure, but there is no evidence for all this. Respiratory distress is rare in previously healthy patients dying during the first two or three days after injury provided that cerebral and thoracic injuries are absent, that blood loss has been corrected by transfusion and that cerebral fat embolism is not present. Respiratory symptoms and complications such as cough, dyspnoea, bronchitis, pneumonia, atelectasis, and infarction are as frequent in injured patients (without head or chest injury) subsequently found to have few or moderate pulmonary emboli as they are in injured patients with numerous pulmonary fat emboli. Records at the Birmingham Accident Hospital show that 25 per cent of patients dying with slight or moderate embolism and 15 per cent of those dying with severe embolism had had respiratory symptoms. There is no evidence of significant blockage of the pulmonary arterial tree, venous engorgement does not occur during the rapid transfusion of patients with multiple fractures or in those with systemic embolism and, in them, the blood pressure does not fall even though there is respiratory distress. Other patients with systemic embolism do not have respiratory symptoms even though heavy pulmonary embolism has probably occurred (see below). Therefore pulmonary embolism is rarely responsible for cardiorespiratory embarrassment. The absence of symptoms must be related to the large functional reserves and enormous capillary bed within the lungs and perhaps to the fluid nature of the emboli which may permit incomplete or intermittent blockage of individual capillaries. When the emboli in a frozen section of lung are stained with a red dye and observed with a red light source, some erythrocytes are often seen between them and the capillary wall.

Morbid anatomy

At autopsy fat embolism is often associated with pulmonary congestion, oedema, small haemorrhages and foci of collapse said to result from capillary and arteriolar

blockage and endothelial anoxia. This is a fallacy of association because similar changes are not uncommon in all injured patients in those dying with few pulmonary emboli and in those with many emboli. Scully (1956) used the weight of the lungs as an index of congestion and oedema and found no correlation with the histological degree of embolism. The lung changes in fatally injured patients must be due to causes other than embolism. Thoracic and cerebral trauma are prone to produce pulmonary congestion and oedema whilst the ever present fallacy of terminal or agonal effects may at times be responsible. Similar changes in those with cerebral fat embolism may be of neurogenic origin.

Lung haemorrhage in certain patients dying after clinical embolism led Harris, Perrett and MacLachlin (1939) to suppose that it resulted from an irritating substance in the fat globules. Haemorrhagic exudation in the lungs of experimental animals was produced by small amounts of hydrolysed human fat the minimum lethal dose of which was only a fraction of that of neutral fat. Other workers confirmed that the intravenous injection of fatty acids and soaps including oleic acid and linoleic acid was toxic to dogs and rabbits in relatively small doses and produced a considerable haemorrhagic pulmonary oedema (Jefferson and Necheles, 1948; Peltier 1956c) but this does not mean that lipase hydrolysis of fat globules occurs or is responsible for the haemorrhagic changes in the lungs sometimes associated with fat emboli. Fatty acids have not been demonstrated histochemically and fat emboli give the reaction of neutral fat when stained by Nile blue sulphate and other dyes (Warren 1946; Denman and Gragg 1948; Sevitt 1956).

Experimental pulmonary fat embolism

Intravenous injection of sufficient neutral fat or mineral oil into experimental animals produces acute respiratory distress, venous congestion and even death, but the maximum tolerable and minimal lethal doses are relatively high and are usually between 0.5 and 2 g. per kg. body weight (Lehman and Moore 1927; Harris, Perrett and MacLachlin 1939; Scuderi 1941). The amount of intravenous fat necessary to kill the average adult could be 70 g. or more assuming that man is as susceptible as animals, but Armin and Grant (1951) showed that the extractable fat in the lungs of patients with gross embolism is only a fraction of this. Moreover the lungs of animals given a lethal dose of fat show a much greater degree of fat embolism than is ever seen in man (Scriba, 1880; Cohnheim 1889) and according to Armin and Grant (1951) the intravenous injection of only 0.15 ml. of fat per kg. into rabbits produced a histological appearance equal to or greater than that seen in gross pulmonary embolism in man but it did not produce symptoms. This dose is equivalent to only 10 ml. for the average adult.

Thus the amount of fat reaching the lungs of patients with heavy embolism seems unlikely to produce clinical effects but against this it has been claimed that serious effects may be produced if the patient is also suffering from shock or blood loss (Killian 1931). Gold and Loeffler (1923) showed that in cats injection of oil prolonged the shock produced by injuring the intestines or compressing the inferior vena cava, but the occurrence of asphyxia and dyspnoea in the non-shocked controls indicated that the dose of fat was about the maximum that could be tolerated. Both Green and Stoner (1950) and Whiteley (1954) found that the minimum lethal dose of fat for rats was reduced when adenosine triphosphate was given or when the animals were subjected to sublethal periods of limb ischaemia,

SYSTEMIC FAT EMBOLISM

whilst Harman and Ragaz (1950) found that the mortality following the injection of fat into rabbits was increased by dehydration or tourniquet-shock. In all these experiments the amount of fat injected was much greater than the dose necessary to produce a degree of embolism comparable to gross embolism in man. When this dose (0.15 ml per kg.) was given to rabbits also subjected to considerable haemorrhage it did not add to the illness or to the mortality nor did the injection of fat into bled animals hinder the beneficial response to blood transfusion (Armin and Grant 1951).

It seems reasonable to conclude that gross pulmonary embolism after injury is unlikely to cause symptoms or produce lung changes in previously healthy subjects and that it is not responsible for death even in patients with shock and haemorrhage. The possibility that respiratory symptoms in those with diseased lungs or a poor myocardium may be aggravated cannot be excluded.

SYSTEMIC FAT EMBOLISM

Systemic fat embolism can undoubtedly produce symptoms and cause death through involvement of the brain. Clinical diagnosis is often difficult because fulminating, incomplete and mild clinical syndromes are not uncommon.

Distribution of emboli

The route of access to the left side of the heart is not known with certainty but the fluid emboli must either make their way through the pulmonary capillaries or by pass this route. Patent foramina ovale were not found in the hearts of a small number of cases dying with systemic (cerebral) fat embolism (Sevitt, 1956) and there is no evidence for the suggestion that the bronchopulmonary venous shunt becomes operative. Thus the pulmonary emboli are probably the immediate source of the systemic embolism.

The distribution of the emboli is dependent on the distribution of the cardiac output and on peculiarities of blood supply within organs. Normally about 25 per cent of the cardiac output goes to the brain and a similar amount to both kidneys so that the density of emboli in these is likely to be high. A post mortem diagnosis of systemic embolism is most easily made by examining the glomeruli for embolic fat because all the cortical blood passes through the glomeruli. Glomerular emboli will be present even when the renal blood flow is reduced following severe injury but the number will be less. They are not infrequently found even when cerebral emboli are scanty or absent. Occasionally no glomerular emboli are seen in patients dying with cerebral embolism and their absence must be attributed to prolonged severe or complete renal ischaemia. Proximal tubular necrosis is often present. Glomerular embolism is not related to traumatic uraemia or renal tubular necrosis whilst cardiac function is unlikely to be affected by the small number of coronary emboli present. On the other hand, the brain cannot withstand blockage of some of its vessels without disturbance of function. Fat emboli are liable to produce minute haemorrhagic and ischaemic infarcts in the cerebral white matter because the arterioles are almost end arteries, but the density of emboli is greater in the capillary network of the cortex. The number of brain emboli is often relatively few compared with the number in the lungs even in

FAT EMBOLISM

patients dying of cerebral embolism. Nevertheless cerebral embolism is potentially grave in contrast to pulmonary and renal embolism which have little or no functional significance.

Relationship to pulmonary fat embolism and severity of injury

Histologically systemic fat embolism is less common than the pulmonary form with which it is always associated. In a series at the Birmingham Accident Hospital 24 per cent of 100 patients had systemic embolism. 89 per cent had pulmonary embolism and all the patients with systemic emboli had pulmonary emboli. The likelihood of systemic embolism is mainly determined by the degree of pulmonary embolism and the severity of bony injury. In the above series systemic embolism was found in 59 per cent of the 32 patients with numerous pulmonary emboli and in only 9 per cent of the 57 patients with few or moderate numbers of emboli. Systemic embolism occurred in 45 per cent of the patients with severe bony injuries but only in 7 per cent of those with mild or moderate bony injuries (Table II). Larger numbers of systemic emboli are likely in patients with heavy pulmonary embolism: in one third of the cases systemic embolism was relatively heavy and all of these had multiple fractures and gross pulmonary embolism.

TABLE II
FREQUENCY OF SYSTEMIC FAT EMBOLISM AFTER INJURY

Series	Number of cases studied	Types of injury	Frequency of systemic fat embolism		
			Total	Clinically significant cerebral forms	Main cause of death
Clinical series					
Wilson And Salisbury (1944)	1,000	Miscellaneous battle casualties		8.8 per cent	0.6 per cent
	119	Battle casualties with fractures of long bones		5.8 per cent	4.2 per cent
Newman (1948)	89	Fractures of long bones		6.0 per cent	3.0 per cent
Musselman, Glas and Grekin (1952)	109	Miscellaneous civilian casualties	52 per cent ¹	14.0 per cent	5.5 per cent
Grant and Reeve (1951)	230	Limb injuries		3.0 per cent	2.1 per cent
	80	Abdominal injuries		0.0 per cent	0.0 per cent
Autopsy series					
Grant and Reeve (1951)	25	Fatal wartime limb injuries	52 per cent	20.0 per cent	
	30	Abdominal injuries	27 per cent	0.0 per cent	
Scully (1956)	110	Fatal battle casualties	17 per cent		1.0 per cent
Birmingham Accident Hospital (Sevitt 1957)	56	Mild or moderate bony injuries	7 per cent	1.8 per cent	1.8 per cent
Emson 1958)	44	Multiple fractures	45 per cent	13.6 per cent	11.3 per cent
	100	Total	24 per cent	7.0 per cent	4.0 per cent

¹ Determined by presence of fat in the urine.

Cases of systemic fat embolism may be divided into three main groups

- (a) *Those diagnosed on clinical grounds (Table II) some of whom die and the diagnosis is confirmed at autopsy.*—This group is often reported as a separate series. The overall incidence reported by Wilson and Salisbury (1944) was 0.8 per cent among 1 000 miscellaneous casualties but this becomes 6 per cent when only patients with fractures are considered. A similar figure was found by Newman (1948) among patients with injuries to long bones, whilst Godlee and Williams (1911) described 3 fatalities (16 per cent) from this cause among 19 patients injured in a railway accident, 18 of whom had fractures of one or both lower limbs. The clinical incidence in Grant and Reeves (1951) wartime series was 3 per cent among patients with limb injuries and 0 per cent among those with abdominal injuries. The frequency of 14 per cent reported by Musselman, Glas and Grekin (1952) was based on a different form of assessment, unexplained cerebral symptoms combined with fat in the urine. Among the factors which influence the clinical frequency of cases are the number of severely injured patients in the series and the criteria of diagnosis.
- (b) *Cases unsuspected during life among those who die after injury (Table II) in whom histological observation reveals systemic fat embolism.*—Diagnosis is based on the demonstration of fat emboli in frozen sections of the brain and kidneys stained with fat soluble dyes. This group may be subdivided into those who had cerebral and other symptoms consistent with fat embolism, and those without significant symptoms. Grant and Reeves (1951) found histological systemic embolism in 13 out of 25 cases (52 per cent) with fatal limb injuries of whom 5 had had clinical symptoms and in 27 per cent of patients dying after abdominal injury but fat embolism was not considered clinically important in these. Scully (1956) found an incidence of 17 per cent among 110 fatal battle casualties but considered that only 1 death resulted from fat embolism. In the series at the Birmingham Accident Hospital the histological incidence of systemic embolism was 24 per cent in 7 per cent symptoms consistent with cerebral fat embolism had been present and in 4 per cent this was considered the main cause of death. These were fulminating cases of cerebral embolism. Coma developed within 12–24 hours of injury and was sometimes preceded by convulsions. Some patients failed to recover consciousness after general anaesthesia.
- (c) *Mild clinically unsuspected cases who survive.*—The frequency is difficult to ascertain but the author's observations and those of his colleagues suggest that it is more common than are cases with obvious symptoms. Some patients develop a fine petechial eruption confirmed by biopsy as due to fat emboli but general symptoms, if present, are only slight. This suggests that other mild cases occur without the petechial rash. Musselman, Glas and Grekin (1952) found that 52 per cent of 109 injured patients had fat in the urine but only a minority had significant symptoms.

Thus the differences between the reported estimates depend on the nature of the series studied, whether clinical or autopsy and within each series on the nature of the injuries, particularly the presence and multiplicity of fractures and the criteria of diagnosis. A series of patients with different degrees of severity of fractures and the cerebral embolism is more frequent than are clinically diagnosed cases. Some of the former are clinically fulminating and others appear to be subclinical. Fulminating cases are important because death may be attributed to the cerebral circulation whilst subclinical cases indicate that the entry of fat globules into the cerebral circulation can sometimes be tolerable. A closer examination of the central nervous system

in injured subjects including perhaps electroencephalography, might reveal other wise undiagnosable cases

CLINICAL CLASSIFICATION AND DIAGNOSIS

The classification into pulmonary cerebral and cardiac forms must be abandoned because the symptoms of fat embolism are cerebral in origin. The fall in blood pressure said to have been part of the cardiac syndrome is not seen in adequately transfused cases developing systemic embolism and probably resulted from untreated oligaemic shock. Cerebral symptoms are dangerous and respiratory symptoms tachycardia, and pyrexia are probably neurogenic in origin. Failure to recognize variations in the clinical picture has been partly responsible for difficulties in clinical diagnosis and for the widely divergent views as to prognosis. Taking these into account fat embolism may be classified clinically into (a) fulminating, rapidly fatal cases (b) the classical or complete syndrome and (c) incomplete and partial syndromes including mild cases

Fulminating cases

As already noted, recognition of these cases has been brought about by correlating the appearance of unexpected and unexplained cerebral symptoms in patients dying within a few days of injury with the presence of cerebral fat emboli at autopsy. The injuries are generally severe multiple fractures are usually present and death occurs 1-3 days after injury even though haemorrhage and oligaemia have been adequately combated by early and efficient blood transfusion. The patient is initially conscious but becomes stuporose and then comatose sometimes within a few hours of injury and generally within 24-48 hours. The onset of coma may be masked by general anaesthesia for emergency surgery but consciousness does not return. Stupor or coma may be preceded or accompanied by epileptiform or choreo-athetoid movements which may be focal unilateral or generalized. Sometimes collapse during resuscitation or failure to maintain a beneficial clinical effect after blood transfusion may dominate the picture cerebral fat embolism is one of the causes of "irreversible shock". Fulminating fat embolism is difficult to diagnose. Clinically the death may be mistaken for the delayed effects of a head injury (which may also be present), for an anaesthetic misadventure or for other causes of coma. The petechial eruption is absent presumably because death occurs before it can develop.

Classical syndrome

The complete syndrome is characterized by pyrexia tachycardia, respiratory symptoms, major cerebral and neurological features particularly delirium and coma, and a characteristic petechial eruption which is of considerable diagnostic importance (Fig. 57). The first symptoms generally develop within 24 hours of the accident and not about the third day as is commonly stated. In the records of 70 published reports and cases observed personally symptoms were present in 23 per cent of cases within 12 hours of injury in 66 per cent within 24 hours, in 80 per cent within 36 hours and in over 90 per cent within 48 hours. The development of symptoms during the 'shock period' is important because their significance may

CLINICAL CLASSIFICATION AND DIAGNOSIS

be overshadowed until the disorder of consciousness is serious or the appearance of the rash focuses the attention of the clinician

The onset is often sudden the temperature rises often to 102-103 F or even higher and the heart quickens to 100-120 beats per minute Both cerebral and respiratory symptoms develop either simultaneously or at different times and one type may predominate Restlessness delirium and insomnia (occasionally mistaken for delirium tremens) often precedes or accompanies mental confusion or stupor and the latter may deepen into coma. Sometimes mental changes improve or disappear and then recur suggesting intermittent embolism of the brain Other possible symptoms include incontinence convulsions disturbances of papillary reflexes, spasticity increased or loss of deep reflexes, and paralysis of a limb or of muscle groups



FIG 57 —The petechial eruption of systemic fat embolism

Respiratory distress is manifest by dyspnoea polypnoea, possibly cyanosis, and tightness in the chest. Moist sounds in the lungs cough and sputum, the latter possibly blood tinged may be present. The blood pressure does not fall unless oligaemia was untreated or until a terminal cardiac failure supervenes and the jugular veins are not distended. Rarely a patient develops distension of the neck veins during the course of blood transfusion for post traumatic oligaemia and this might be the result of pulmonary fat embolism

The petechial eruption develops on the second or third day after injury and is generally preceded by other symptoms. It first appears on the fronts of the shoulders root of the neck, and the upper part of the chest. The rash may be restricted to these areas or may extend over the arms upper abdomen and thighs. In most cases the petechiae are fine red spots but sometimes they are small macules or a mixture of spots and macules. They appear in crops over the course of 1-2 days each crop fading within a day or so. The uninitiated may mistake the combination

of pyrexia and petechial eruption for evidence of septicaemia. Petechiae have also been reported in the mouth, palate, conjunctivae and retinae but they are not constant.

Incomplete and partial syndromes

There are many proven cases in which characteristic features are absent. The author's series included 12 cases of proven fat embolism as indicated by the characteristic rash and the finding of embolic fat globules in a biopsy of affected skin. Their symptomatology fell into four groups: (a) fever, tachycardia, some mental confusion and a petechial rash but without other cerebral neurological or respiratory symptoms; in 1 patient mental symptoms were confined to retrograde amnesia; (b) pyrexia, tachycardia, a variable degree of dyspnoea and a petechial rash but without mental or neurological symptoms; (c) pyrexia, tachycardia, and a rash without mental neurological or respiratory symptoms; and (d) slight fever and a definite rash without tachycardia, respiratory or mental symptoms. Nearly all ran a mild course and all survived. The rash drew attention to fat embolism which otherwise would have remained undiagnosed in most cases.

Many cases of fat embolism without a rash occur in addition to the fulminating ones already described. Some may run a severe course with major cerebral and respiratory symptoms; in others cerebral or respiratory symptoms or both may be absent. It is particularly in the patients without a rash that laboratory aids to diagnosis would be most useful.

Possible aids to diagnosis

Unfortunately no reliable and simple test has yet been developed and diagnosis remains largely a matter of clinical acumen.

Fat in urine

Diagnosis of renal and hence systemic embolism might be made by this test but most observers find it unreliable; in the author's experience most specimens of urine from patients diagnosed as having systemic embolism do not contain fat. Negative results may be due to incomplete emptying of the bladder since fat floats in urine. On the other hand, Musselman, Glas and Grekin (1952) found urinary fat in 52 per cent of 109 injured patients and in 12 per cent of 50 uninjured subjects but they examined 15 consecutive 24-hour specimens of urine in each patient. If the test is used scrupulous care must be taken to prevent only contamination of the specimen and to ensure complete evacuation of the bladder.

Fat globules in blood

Microscopy of the blood for globules of fat was suggested by Scuderi (1941) and Pelletier (1954) developed a test based on the staining of fat in plasma by the water soluble fluorochrome dye phosphine 3R. By fluorescent microscopy he found fat globules in the plasma of 55 per cent of patients undergoing orthopaedic operations and later (Pelletier 1956a) reported positive results in 80 per cent of 44 patients following bone operations but in only one patient did the classical signs of fat embolism occur. Further work is obviously required particularly on the concentration of fat globules and on the relationship of positive tests to clinical signs and pathological evidence of fat embolism.

PROPHYLAXIS AND TREATMENT

Renal biopsy

Needle biopsy of the kidney should be considered in special cases. The author has found it useful in diagnosing systemic embolism in obscure cases of post traumatic coma particularly when craniotomy is being considered for a possible intracranial haemorrhage. It should not be lightly undertaken but using the Vim-Silverman needle the technique is relatively safe and is not difficult. The core of renal tissue should be gently teased flattened between two slides and stained for fat by any of the fat soluble dyes. The finding of glomerular emboli indicates systemic embolism and by inference cerebral embolism.

Other tests

The finding in sputum of fat globules larger than $10-12\ \mu$ in diameter is of limited value and at most indicates pulmonary embolism. The radiological appearance of fluffy or "snow-storm" lung fields is inconstant and this shadowy evidence may be due to other causes.

PROPHYLAXIS AND TREATMENT

Prophylaxis

Little can be added to the old advice of early gentle reduction and immobilization of fractures to reduce dislodgment of fat and the use of a tourniquet when possible during orthopaedic operations. The intramedullary nailing of long bones is said to be a special hazard and fatal cases of fat embolism have been reported by Haebler (1949) Böhler and Böhler (1949) Peltier (1952) and others. The hammering of the nail is said to raise the intramedullary pressure above the systolic blood pressure (Kuntzschner 1940) and to force fat into veins. Surgeons have therefore been advised not to use a solid nail and to pause between the hammer blows.

Treatment

Treatment is still empirical and symptomatic. Respiratory distress when present is often alleviated by the use of an oxygen tent. Sedatives may be essential when the patient is delirious or very restless.

Fat dispersing and emulsifying agents

Specific drug therapy has not yet been established but certain lines of research warrant further investigation. Emulsifying and fat-dispersing agents have been tried in animals subjected to fat embolism with the purpose of clearing the blood lungs and other organs of fat globules. Some substances were obviously ineffective or even dangerous including sodium lauryl alcohol sulphonate and sodium dehydrocholate assessed by Scuderi (1941), Tween 80 investigated by Glas, Grekin and Musselman (1953) sodium carbonate, adrenaline and inhalation of ether. Therapeutic value was claimed for intravenous choline chloride by Monson and Dennis (1949) who treated fat embolism produced in dogs by curettage of the bone marrow but their claim is doubtful because the degree of lung embolism was apparently not affected. Investigation of the claims made for alcohol-glucose solutions by Hermann (1933 1952) is required. Fat embolism, produced in hares and dogs by an intravenous injection of fat (1 ml per kg.) was treated with a

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solution consisting of 3 parts of ethyl alcohol mixed with 7 parts of 25 per cent glucose solution given intravenously in a dose of 5 ml per kg. Injection of the mixture relieved dyspnoea and cyanosis and the lung vessels of animals given 3 injections at 12 hourly intervals were found to be almost free of fat. The therapeutic dose was about half the narcotic dose and a quarter of the lethal dose.

The ability of heparin to clear lipaemic plasma (Hahn 1943) suggests that it might be of value in fat embolism. However heparin treatment of rats given rat fat intravenously was found to increase the mortality and accelerate death (Gardner and Harrison 1957). It was concluded that a clinical trial would not be warranted. Nevertheless 3 patients suffering from systemic and cerebral fat embolism were treated in this way by Sage and Tudor (1958) for 2-3 days without ill effect and the patients recovered.

REFERENCES

- Armin, J. and Grant, R. T. (1951) *Clin Sci* 10, 441.
 Belli, C. J. (1942) *Surgery* 12, 251.
 Böhler L. and Böhler J. (1949) *J Bone Ji Surg.*, 31A, 2951.
 Caldwell G. T. and Huber H. L. (1917). *Surg Gynec Obstet.*, 25 650.
 Cohnheim J. (1889) *Lectures in General Pathology* London.
 Denman, F. R., and Gragg, L. (1948) *Arch Surg Chicago* 57 325.
 Emson, H. E. (1958) *J clin. Path.* 11 28.
 Frazer A. C., Elkes, J. J., Sammons, H. G. Govan, A. D. T. and Cooke, W. T. (1945) *Lancet* 1 457.
 Gardner A. M. N., and Harrison, M. H. M. (1957) *J Bone Ji Surg.* 39B, 538.
 Gauss, H. (1916) *Arch Intern. Med.* 18, 76.
 — (1924) *Arch Surg., Chicago* 9 593.
 Glas, W. W. Grekin, T. D. and Musselman, M. M. (1953) *Amer J Surg* 85, 363.
 Godlee R. J. and Williams, G. E. O. (1911). *Lancet* 1 1062.
 Gold, E. and Loeffler E. (1923) *Z. ges. exp. Med.*, 38, 155.
 Grant, R. T. and Reeve, E. B. (1951) Observations on the General Effects of Injury in Man." *Spec. Rep. Ser. med. Res. Coun., Lond* No 277 London H.M. Stationery Office.
 Green, H. N., and Stoner H. B. (1950). *Biological Actions of the Adenine Nucleotides* London Lewis.
 Gröndahl, N. B. (1911). *Dtsch. Z. Chir.*, 111 56.
 Haebler C. (1949) *Nat. med. Bull. Wash.* 49, 423.
 Hahn, P. F. (1943). *Science* 98, 19.
 Harman, J. W., and Ragaz, F. J. (1950) *Amer J Path.*, 26, 551.
 Harris, R. J., Perrett, T. S. and MacLachlin, A. (1939) *Ann. Surg.*, 110, 1095.
 Hermann, L. G. (1933) *Proc. Soc. exp. Biol* 30, 558.
 — (1952) *Arch Surg., Chicago* 65, 556.
 Holm (1876) *Beitr. z. Lehre v.d. Fett-embolie*, München (quoted by Armin and Grant 1951).
 Jefferson, N. C. and Necheles, H. (1948) *Proc. Soc. exp. Biol* 68, 248.
 Killian, H. (1931) *Dtsch. Z. Chir.* 231 97.
 Künzschner G. (1940) *Klin. ther. Wschr.*, 19 6.
 Lehman, E. P. and Moore, R. M. (1927) *Arch. Surg. Chicago* 14 621.
 Lubarsch O. (1893). *Fortschr. Med.*, 11 805.
 Mallory T. B. (1952). *Physiologic Effects of Wounds.* U.S. Army Report # 283 Washington.
 Meek, K. (1892) *Beitr. klin. Chir.*, 8, 421.
 Miloslavitch E. L. (1930). *Wis. med J.* 29 139.
 Monson, E. M., and Dennis, C. (1949) *Proc. Soc. exper. Biol* 70, 330.
 Musselman, M. M., Glas, W. W. and Grekin, T. D. (1952). *Arch Surg Chicago* 65, 551.
 Newman P. H. (1948) *J Bone Ji Surg.* 30B, 290.
 Payr E. (1900). *Z. orthopaed. Chir.* 7 338.

REFERENCES

- Pettler L. F. (1952) *Surgery* 32, 719
- (1954). *Ibid.*, 36, 198
- (1956a) *J Bone Jt Surg.*, 38A, 835
- (1956b). *Surgery* 40 657
- (1956c) *Ibid* 40, 665
- Reiner M (1907). *Münch med Wschr* 54, 2004
- Robb-Smith, A. H. T. (1941). *Lancet* 1 135
- Sage, R. H., and Tudor R. W. (1958) *Brit med J* 1 1160
- Scriba, J. (1880). *Dtsch Chir.*, 12, 118
- Scuderi C. S. (1934). *Int surg Dig.*, 18, 195
- (1938). *Arch Surg.*, Chicago 36, 614
- (1941). *Surg Gynec Obstet.*, 72, 732
- Scully R. E. (1956) *Amer J Path.*, 32, 379
- Sevitt, S. (1956) Unpublished observations.
- (1957). In Proceedings of the Pathological Society of Great Britain and Ireland 1956. Abstracted in *Aed Tijdschr Geneesk.*, 1522.
- Sheynis, M. I. (1931). *Arch. Pat.*, 13, 103
- Vance, B. M. (1931). *Arch Surg.*, 23, 426.
- (1934). *Amer J Surg.*, 26, 27
- Wagner E. (1862). *Arch. f. Heilk. Bd.*, 3, 359
- Warren, S. (1946) *Amer J Path.*, 22, 69
- Warthin, A. S. (1913). *Int Clin.*, 4, 171
- Whiteley H. J. (1954) *J Path. Bact.*, 67 521
- Wilson, J. V., and Salisbury C. V. (1944) *Brit J Surg* 31 384
- Young, J. S. and Griffith H. D. (1950) *J Path Bact.*, 62, 293
- Zenker F. A. (1862). *Beitrage zu normalen und pathologischen Anatomie der Lungen* Dresden.

CHAPTER 15

ABDOMINAL AND CHEST INJURIES

RUSCOE CLARKE AND A. L. D. ABREU

ABDOMINAL INJURIES

IN Great Britain the ratio of penetrating to closed abdominal injuries requiring surgery is about 1 to 5 in contrast to many countries where knife and revolver wounds are common and to war experience where penetrating wounds predominate. The predominance of closed injuries is important because penetrating wounds have received greater attention in the literature and there is a tendency to generalize from these to the rather different problems presented by civilian closed injuries.

PENETRATING WOUNDS

Civilian wounds of abdominal organs resulting from penetration of the abdominal or thoracic parietes are usually straightforward but must be treated early. Any penetrating wound of the trunk which could possibly have reached the peritoneal cavity must be explored. If there remains the slightest doubt after exploration of the wound track the abdominal cavity must be opened, and it should be remembered that the direct course of whatever penetrates may become tortuous when the patient adopts a different position.

More severe injuries in civilian life result from penetration of the perineum, rectum or vagina from spikes, broom handles or other long objects. Again, there should be no hesitation in carrying out full exploration, including laparotomy. If there is still any doubt about the integrity of the rectum or lower urinary passages, a colostomy or suprapubic cystotomy is indicated. Partial damage to the bladder, urethra or rectum or damage to their supporting tissues can lead to late sloughing and the insidious or fulminating development of cellulitis. Colostomy, cystotomy or both can prevent distension of the injured viscus, reduce the danger of delayed rupture and minimize the ill effects should rupture occur.

The greatest danger from penetrating wounds exists (1) when the wound is so small that there is a temptation to neglect it and (2) when other injuries are so severe that it is overlooked. The absence of symptoms or local signs never excludes the possibility of a dangerous lesion of the gut.

CLOSED INJURIES

The pattern of abdominal injuries is changing with the growing frequency of road injuries involving high velocities of impact. Sometimes the history is of a blow on the abdominal region and the clinical problem simply that of excluding an intra-abdominal lesion requiring surgery. Occasionally such local injuries are associated with massive haemorrhage and present a major problem of resuscitation and surgical control of bleeding.

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Increasingly however abdominal injuries are associated with more general damage to the trunk or body following vehicle accidents or falls from heights. Diagnosis and treatment are complicated by the complexity and severity of the associated injuries. With the milder injuries there is usually time for careful assessment but with major and multiple injuries treatment has to be started immediately. The decision to explore the abdomen may be extremely difficult in a severely injured patient in whom an unnecessary laparotomy might tip the scales unfavourably.

No advance in the diagnosis of closed abdominal injuries has invalidated the proposition that laparotomy must often be performed on mere suspicion that a dangerous internal lesion has occurred. It is a misconception to approach the clinical diagnosis in terms of individual organs each with its specific signs and symptoms of injury. The diagnosis of abdominal disease is based on evidence of infection, peritoneal irritation, intestinal obstruction, a mass or bleeding, but in the most easily treatable stages of abdominal injury one often has no more to go on than evidence of minor peritoneal irritation, perhaps signs of parietal injury. Sometimes the general condition of the patient. The internal lesion may not be located at the site of injury pain or physical signs. Peritoneal irritation caused by moderate haemorrhage from a ruptured spleen or liver may not be clinically distinguishable from that caused by a perforated hollow viscus, whilst a dangerous split of the outer coats of the gut may be associated with a minimum of signs.

When there are also other injuries an important abdominal lesion can be overlooked even on careful clinical investigation. Symptoms and signs may be obscured by painful lesions elsewhere or inhibited by an associated head injury. On the other hand, abdominal signs can be produced by injury to the abdominal wall, fractures of the ribs, spine or pelvis, and by thoracic injury without bony damage. Sometimes no clear explanation of such signs is found even at laparotomy. Although operation must often be performed on mere suspicion it should normally follow a period of thoughtful clinical investigation and observation. A number of clinical features warrant further discussion.

History

The patient may give a history of abdominal injury but occasionally the history is such that an abdominal lesion is not even considered. Rupture of the spleen has been known to follow an injury to the right thigh, a twist while dropping a few feet to the ground and other more or less trivial injuries remote from the site of trouble. Following severe falls from a height or a road accident, the presence of injuries elsewhere does not exclude the need to consider the abdomen. The absence of pain is not significant. Where associated injuries are relatively severe the patient is likely to be kept under observation but when a patient walks into hospital following a car or cycle accident complaining of a specific minor lesion somewhere else, the abdomen may not be examined until the patient collapses with perforation of a viscus or delayed haemorrhage. Even then, the patient may deny any recollection of causal injury.

General condition

Except when there is severe abdominal bleeding, the patient's general condition is of little aid in making a diagnosis. A penetration of the gut with a small leak can

produce a state of shock with a low blood pressure pallor cold extremities, a rapid or slow pulse sweating, vomiting and anxiety. Improvement may take place without treatment in the absence of gross peritoneal soiling or excessive fluid loss, but even this does not provide a clear differentiation from haemorrhage. The individual with a suspected abdominal lesion who has collapsed or fainted at any stage should be watched with particular care.

Clinical examination

The patient with severe abdominal pain and guarding, thoracic respiration, shoulder tip pain and so forth, is clearly suspect. The chief consideration is often not so much whether an abdominal lesion is present but whether the signs and symptoms can be sufficiently explained by injury to the chest, spine or pelvis. X ray examination may give clues that visceral damage or abdominal bleeding has occurred but cannot exclude them. A more difficult problem exists where symptoms are mild and physical signs indefinite. In the early stages of acute appendicitis the key physical sign is local tenderness over the appendix. In abdominal injuries of doubtful significance the key physical sign is reflex muscle contraction provoked by gentle pressure rather than by deep palpation. This is particularly valuable in detecting the spread of physical signs from one region to the whole abdomen. With chest injuries or injuries confined to the renal or perirenal tissues, guarding in this sense and tenderness may be sharply confined to the upper abdomen or the flank and one side of the rectus abdominis muscle. The presence or development of minimal guarding in the rest of the abdominal area should raise strong suspicions of an intraperitoneal lesion. Hyperaesthesia is of less value after recent injury than when infection is suspected. Auscultation of the abdomen is useful but often misleading. Absence of bowel sounds can be associated with retroperitoneal haemorrhage or bruising of the mesentery whilst normal or exaggerated bowel sounds may be present with a perforated gut. Rectal examination is usually inconclusive unless rectal bleeding is found.

Damage to the abdominal wall rather than to its contents may be suspected from visible bruising and an increase in local pain when the muscles are contracted. The disappearance of tenderness when the muscles are held contracted tends to favour injured abdominal contents rather than an abdominal wall lesion, but an abdominal wall lesion in no way precludes a lesion inside.

Percussion is not of much value unless a considerable haematoma has developed shifting dullness usually accompanies other signs that make its detection unnecessary and the disturbance of demonstrating it may be harmful.

Repeated examination is more valuable than a single test and is often most informative if the patient is left alone between examinations. Following a period of rest, the first person to carry out an abdominal examination is likely to make the most valuable observations. The more frequently palpation is carried out the more likely are equivocal signs to be elicited.

Observation includes regular charting of pulse rate, respiration rate and blood pressure but instruction should be given so that any change in symptoms or signs leads to prompt reassessment. A rising pulse rate, a falling blood pressure, or increasing pallor may indicate progressive bleeding. Increasing pain is important and vomiting should arouse suspicion. The evaluation of general signs is particu-

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larly difficult when other injuries are associated with bleeding. At times it may be necessary to transfuse an amount of blood sufficient to cover estimated losses from known sources and then to stop, so as to establish a new base line for further observation.

Coffee-ground vomiting may follow multiple injuries without an abdominal lesion but is always an extra factor favouring laparotomy. The urine should be examined. Haematuria means that the urinary tract has probably been injured although its absence does not preclude significant recent damage with an obstructed pelvis or torn ureter. Normal urine and a normal pyelogram have been seen in the presence of a deep cleft in the kidney with extensive perinephric haemorrhage (Fig. 58)



(a)



(b)

FIG 58—A deep tear of the right kidney with normal renal function (a) the kidney (b) the intravenous pyelogram

Radiology

The presence of free gas in the peritoneal cavity is important. It may be demonstrated either under the diaphragm with the patient sitting or in the flank with the patient lying on his side. After injury free gas in the peritoneum or surgical emphysema in the retroperitoneal tissues means that perforation of the gut has occurred. A splenic haematoma may sometimes be revealed because it distorts the gastric air bubble the shadow of a large perinephric haemorrhage will obscure the edge of the psoas muscle. When haematuria is present or renal damage suspected intravenous pyelography should be carried out if time permits. Even following severe oligæmic collapse a good picture can usually be obtained once the blood volume has been restored to near normal. With unilateral renal damage urography will show whether or not there is another functioning kidney. In the

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presence of haematuria, intravenous pyelography is particularly necessary where exploration is indicated for some other injury. The fact that an injured kidney shows no sign of excretion does not necessarily mean that it is irreparably damaged. At least as often it will excrete some of the radio-opaque material and show normal renal pelvis extravasation or incomplete filling of the pelvis suggesting the presence of blood clot or major damage (Fig. 59).



FIG. 59—Clinically this patient had a major rupture of the right kidney. Intravenous pyelography showed the outline of the upper part of the kidney only. The diagnosis of a separated lower pole was made pre-operatively. (a) the kidney. (b) the intravenous pyelogram.

Aspiration

Collier (1954) advocated aspiration of the peritoneal cavity as an aid to diagnosis. A fine-gauge needle is inserted below the costal margin in each flank or occasionally in the iliac fossa. The test was assessed as being 75 per cent accurate in the diagnosis of intra abdominal haemorrhage. It is probably most valuable when the general condition of the patient indicates the existence of severe bleeding in the absence of abdominal signs, particularly in patients with severe head injuries or traumatic tetraplegia. In other conditions the test is not sufficiently accurate and is less useful. Aspiration of blood does not necessarily mean that the risk to life will be increased if laparotomy is postponed, whilst a negative puncture does not exclude a lesion requiring surgery. Very much the same reasoning applies to the suggestion that the risk of laparotomy can be minimized by making a small hole

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and inserting a sponge on a sponge holder. The results of limited exploration may be inconclusive and if it has been already considered that laparotomy carries a deterrent risk, valuable time will have been wasted without a diagnosis being made.

Pre-operative assessment of blood loss

Pre-operative assessment of blood loss can be extremely difficult unless the patient is obviously suffering from massive haemorrhage. A patient with a normal blood pressure and a rigid abdomen may require a transfusion of 6-8 or more pints of blood whilst the patient who is apparently fit may have already lost 4 pints of blood or more. On the other hand patients with similar clinical features may have intestinal lesions with no significant bleeding. The amount of blood cross-matched in advance will depend on the judgment of the surgeon and on the laboratory facilities and quantity of blood available (see Chapter 6). It is wise to err in the direction of over-estimation but if haemorrhage is doubtful pre-operative transfusion should consist of 5-per-cent glucose solution in one fifth normal saline solution the blood being reserved until the abdomen is opened and the diagnosis clarified. If the patient's general condition is serious, transfusion of blood should not be delayed.

Operation

Incision

Even when the site of an abdominal injury is suspected on clinical grounds, complete exploration is necessary and must be done under vision. A vertical incision is advisable, and should be long enough to put both hands inside the abdomen. If the patient is seriously ill and time is all important, a mid-line or paramedian incision gives a quick opening and closing. When there is time a paramedian incision will give a sounder wound. Thorough exploration is assisted by the relaxation obtained with modern anaesthetic agents but the injured abdomen cannot be properly explored with the small gut inside. Particularly in the presence of bleeding it is often wise to allow the whole of the small intestine to prolapse. This is not dangerous with a large incision and does not cause shock with adequate replacement therapy. It allows the small intestine to be examined rapidly loop by loop on each side, and the whole of each flank of the abdominal cavity to be clearly seen with the aid of a retractor under the corresponding half of the abdominal wall. The ruptured spleen is best removed under vision by lifting it forward into the wound after tearing through the posterior peritoneal attachment (Clarke 1954).

Exploration

In the presence of massive bleeding, it can be vitally important to find the source of bleeding with a minimum of delay. This requires a standardized technique of laparotomy. The hand is first placed down to the spleen: most splenic ruptures will be recognized by feel. If the spleen feels normal the hand should examine both lobes of the liver front and back. Damage here can be felt and temporarily packed off while the rest of the abdomen is explored. Tears in the diaphragm can be felt during the above manoeuvres. The next step is to inspect the stomach, small intestine flanks and pelvis, in that order. This will also reveal bleeding from the mesenteries or retroperitoneal damage.

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It is not proposed to give details of the repair of individual injuries but it should be emphasized that when injuries are multiple or severe, control of bleeding and repair of viscera should be done by the simplest possible technique which will ensure haemostasis and healing. The injured spleen is removed the torn liver can sometimes be sutured, occasionally a half-separated segment can be resected. Superficial clefts or raw areas of the liver are best left alone or covered with a gelatin-foam pack. When bleeding cannot be otherwise controlled there is a place for packing with a gauze roll.

Lesions of the left colon often warrant exteriorization. Small lesions of the right colon are better repaired and a caecostomy performed to prevent tension on the suture line. Occasionally a severe closed injury to the right side of the colon will warrant primary right hemicolectomy with ileo-transverse colostomy and exteriorization of the right end of the transverse colon. In any emergency this can be done quickly. Resection of the small intestine is rarely indicated but is safer early after trauma than in the presence of strangulation or infection. There is rarely any need to drain the peritoneal cavity but a drain should be put down to the site of an extraperitoneal rupture of the duodenum or fixed portion of the colon when the latter is not exteriorized. Except after nephrectomy drainage is always necessary when the urinary tract has been damaged.

Post-operative care

Post-operative care does not generally differ in principle from the post-operative care of patients undergoing planned major abdominal surgery for disease. In our experience adequate transfusion makes for early recovery of normal bowel function and early return to normal feeding. When normal feeding is likely to be delayed the intravenous intake must take into account probable fluid and electrolyte losses. Sodium and chloride are generally retained by the kidney so that a reduced excretion of salt is not *per se* an indication for an increased intake. Potassium needs to be given when oral feeding is delayed or loss likely to be great.

Gastric suction is now less often employed after abdominal operations and injuries than it was in the recent past. It remains an invaluable method of preventing abdominal distension and treating early ileus, so allowing an injured section of the gut to heal without gaseous distension, which interferes with its blood supply as well as putting a strain on the suture line.

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By the close of World War II a trend towards a rational and well-grounded approach to the management of severe chest injuries had been established and two main principles had emerged (1) prompt restoration of effectual pulmonary ventilation and of the oxygen-carrying capacity of the blood is life saving and (2) the restoration and maintenance of normal pulmonary function minimizes complications and facilitates treatment of other injuries. The urgent needs, therefore, are the rapid replacement of missing blood, clearance of the airway if obstructed by blood, mucus or pus, and re-expansion of the lungs if compressed by blood or air in the pleural cavity. These measures often correct anoxia and prevent the

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accumulation of carbon dioxide in the blood but oxygen may have to be given. The second stage restoring full lung function calls for a stable efficiently moving chest wall acting on lungs working within a pleural cavity free from trapped air or blood and with no obstruction within the bronchial tree. Treatment is comparable to that of a fracture accurate reduction of displaced tissue maintenance of this by suitable means and the performance of active functional movements under physiotherapeutic supervision.

Traumatic pneumothorax

Open sucking pneumothorax

In civilian practice the open sucking pneumothorax is uncommon but dangerous when it occurs. The experience of war surgery going back to the time of Paré has shown that immediate occlusion of the open wound by a firmly anchored pad or temporary suture is the prime surgical duty. Within minutes of such closure paradoxical movement of the lungs ceases and air passes in and out through the trachea instead of from one lung to another no longer does the shunting of carbon dioxide between the lungs allow a steady rise of that gas in the plasma with consequent violent hyperpnoea and general deterioration. Left undetected this rise in circulating carbon dioxide leads first to a period of respiratory excitation and systemic hypertension, and subsequently to poisoning of the respiratory centre coma and death.

Once the initial distress has been overcome the wound is treated on the accepted lines of wound surgery (see Chapter 8) followed by suture. The decision whether or not to use intercostal water-seal drainage of the hole in the chest wall depends on the state of the underlying lung should this be the site of air leakage, drainage will be required. If drainage is employed it should be adequate, with two intercostal water-sealed tubes to ensure that both air and blood escape steadily and allow the lung to expand fully without delay (Fig. 60).

Closed haemo-pneumothorax

More commonly the pneumothorax is a closed one and is accompanied by a haemothorax usually the lacerated lung will collapse and the air leak will cease. Treatment requires aspiration of air and blood. Experience gained from war casualties has shown that early aspiration is not followed by further haemorrhage. The removal of the blood and air allows the lung to re-expand, and this in turn allows not only re-aeration but also a normal blood flow to be re-established with improvement in the venous return and cardiac output. Following pleural decompression, the general condition usually shows a dramatic improvement. Atelectasis of a lung or a lobe within the air filled pleural cavity may require bronchoscopic aspiration.

Tension pneumothorax

Exceptionally a patient is seen with a tension pneumothorax. This condition, which produces a steadily rising intrapleural pressure as the result of a valvular leak, is usually due to a tear at the base of a pulmonary adhesion (seen commonly



FIG. 60—Adequate drainage of the pleural cavity with two tubes and water seal drainage.

in patients with a "spontaneous" pneumothorax) or injury to a bronchus of moderate size. In either case the tear opens during inspiration and air flows into the pleural cavity; on expiration the tear tends to close and the trapped air cannot escape.

A patient with a tension pneumothorax is gravely ill. The rising pressure in the pleural cavity not only compresses and defunctions the underlying lung, but pushes the mediastinum over, decreasing the efficacy of the opposite lung and hampering the venous return through the venae cavae. The cyanotic pallor and feeble pulse indicate the need for immediate relief, which dramatically follows the institution of closed water-seal drainage. In the first instance this is provided by inserting a large bore needle connected to suitable tubing into the pleural cavity; the tubing is connected to a water-sealed bottle through which the air rapidly bubbles out. If the air drainage so provided is inadequate a catheter is introduced into the pleural space through a trocar and cannula. If large quantities of air continue to escape the surgeon should consider the possibility of a major tear of a bronchus.

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Major bronchial tears

With the increase of high speed accidents a tear of a bronchus is becoming commoner and its possibility needs to be borne in mind. Such injuries are usually the result of a shearing force. Occasionally this also ruptures the arch of the aorta at its junction with the ascending or descending part this is more likely in aircraft crashes because of the tremendous violence involved, but aortic rupture also sometimes occurs in the "steering wheel" type of car injury. Usually the tear is in a main bronchus, commonly the left bronchus. The bronchus may be torn with, or more commonly without a tear of the pulmonary artery. Many of the survivors of this formidable injury have been diagnosed later sometimes years after the accident. If large quantities of air continue to escape into the pleural cavity this injury should be suspected. Radiological examination will show a collapsed lung, which should lead to bronchoscopy the bronchoscope will disclose an area of blood in the region of the tear. The indication for immediate thoracotomy is obvious. These tears are amenable to immediate suture and the apposed bronchial surfaces heal well.

Late cases

In the later cases the patients are referred because clinical and radiological examination has demonstrated an airless lung. At bronchoscopy the main bronchus is seen to end in a mass of fibrous tissue. Even after years of collapse delayed suture of the bronchus can be followed by re-expansion of the lung. In many patients bronchospirrometry and cardiac catheterization have shown that the lung can also regain its function. Such happy results only follow when the lung has remained free from considerable infection if the lung is severely infected pneumonectomy or lobectomy is necessary.

Injury of major vessels

Damage to the aorta in crush injuries is not always fatal and traumatic aneurysm can develop commonly in the region of the left subclavian artery. Today such injuries are amenable to surgery (De Bakey Cooley and Creech 1955). Usually the diagnosis of the damage is delayed and, fortunately surgery may also need to be delayed. The problem is akin to that of arterial haematoma in the limbs, where in the early stages a watching policy may be indicated. If after an initial shrinking the aneurysm begins to enlarge it should be operated on. Either the aneurysm is excised at its neck if sacular in type, or the area of the aorta affected is resected and replaced by an aortic graft. The operation is carried out under hypothermia or with a temporary arrangement whereby the aneurysm is by passed and adequate oxygenation of the spinal cord is maintained during a fairly long operation.

The stove-in chest with paradoxical movement

Following a severe crush injury in which the head as well as the chest and abdomen may be involved severe multiple rib fractures can easily be overlooked. The serious consequences of a stove in chest may not be obvious until 24 hours after the accident. When several ribs in sequence are broken in two or more places

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the stove-in segment is no longer under normal muscular control and respiratory movements tend to affect the lungs in the same way as with an open pneumothorax. The loose segment is sucked in with inspiration as the opposite lung inflates. It is pushed out when the opposite side deflates in expiration thus providing oscillation of gases from one lung to the other with a steady increase in plasma carbon dioxide. The patient may tolerate this without obvious dyspnoea and distress for the first few hours but with the passage of time the rise in plasma carbon dioxide is aggravated by the retention of bronchial secretions as coughing becomes increasingly more painful and ineffective. Inadequate oxygenation is added to carbon-dioxide poisoning. The patient's condition deteriorates.

Tracheotomy

In an attempt (usually successful) to prevent this tracheotomy is being used increasingly for such injuries combined with measures to stabilize the broken floating ribs. In the first stage of treatment the chest wall should be steadied by the application of firm compression. Tracheotomy should be done early if there is any evidence of increasing dyspnoea and coughing.

The benefits of tracheotomy are as follows: (1) it reduces the dead-space air and allows tidal air to be better utilized; (2) it eliminates the resistance offered by the larynx and upper air passages and the effort of the respiratory movements is greatly lessened; (3) it allows bronchial toilet by suction to be carried out more effectively and more often.

When chest injuries are accompanied by head injuries with persisting unconsciousness tracheotomy should be done as early as possible. In trunk injuries in which the chest, and often the abdominal viscera, are seriously damaged, tracheotomy should be done as a first-aid measure when the chest moves paradoxically when there is dyspnoea or cyanosis or when the patient is unable to cough up bronchial mucus.

Tracheotomy should now be regarded as a deliberate method of maintaining a clear airway and not as a last desperate means of restoring it. This being so the artificial airway should be made under operating theatre conditions and only rarely as a dramatic urgency. Most tracheotomies for chest injuries can and should be done deliberately under general anaesthesia. As soon as the anaesthetist has produced light anaesthesia and relaxation (by a relaxant) he passes an intratracheal tube and ventilates the lung fully by hand or machine. The surgeon can then carry out the tracheotomy deliberately using as large a bore tracheotomy tube as possible to facilitate bronchial toilet and suction (Fig. 61). The addition of tracheotomy to the care of severe crush injuries of the chest has been the outstanding advance in treatment in the last few years. The immediate change both mental and physical in the anxious dyspnoeic patient, is more than striking and no patient with a closed injury of the chest, however severe, should be allowed to die without this measure being carried out.

Assisted ventilation with tracheotomy

Usually tracheotomy alone suffices for indicated in cases of associated cerebral injury and respiration maintained in bilaterally paralyzed patients where the respiratory mechanism is defective.

Indicated in patients in whom this measure is and in patients with associated mechanism for normal ventilation by relieving the muscular

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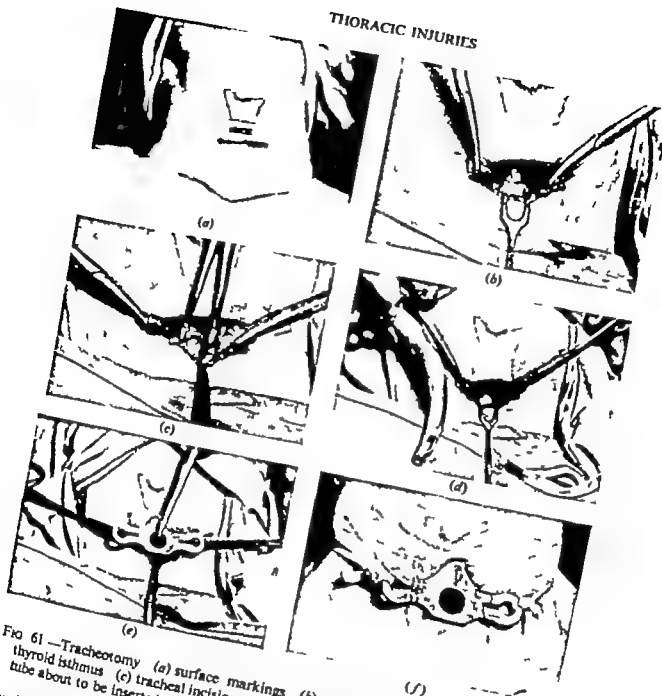


FIG 61 —Tracheotomy (a) surface markings (b) transverse incision and division of thyroid isthmus (c) tracheal incision and intratracheal tube exposed (d) tracheotomy tube about to be inserted (e) bronchial suction (f) tracheotomy fixed in position.

burden of exaggerated but unavailing respiratory efforts. It is then important to provide help by means of breathing machines of which the Beaver and the Smith-Clark types are excellent it would be inappropriate here to discuss the techniques which are essentially those used in the treatment of severe cases of tetanus and poliomyelitis

Drugs

The danger of depressing respiration by injudicious doses of opiates and barbiturates has been mentioned in patients with moderately good ventilation severe

pain must be relieved. Pethidine should be tried before resorting to morphine or Omnopon the sensible relief of pain often aids rather than depresses respiratory efforts. The over-anxious patient is relieved by carefully given doses of chlorpromazine the dangers of this type of drug are well understood today but they can be used if the medical and nursing staff have a sound knowledge of their disadvantages as well as their advantages. Procaine injected locally often relieves the severe pain of rib fractures. Intravenous procaine may severely lower blood pressure and should be used with care, particularly when hypotension is present.

Stabilization of the "flail" chest

When several ribs have been fractured, there is an increasing tendency to rely on restoring normal active movements rather than to rely on the passive stability provided by strapping or plaster fixation. Frequently in compression injuries the ribs are broken at two sites leaving an area of the chest wall to move paradoxically (Fig. 62). This abnormal movement can be checked by direct operative repair.

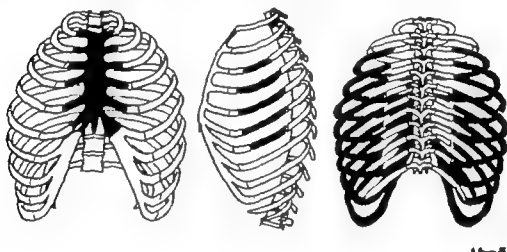


FIG. 62.—Types of segmental injury to the thoracic cage. The floating segments are in solid black (a) anterior (b) lateral (c) posterior (From Proctor and London (1955) by courtesy of the authors and *British Journal of Surgery*.)

sometimes re-aligned fragments can be fixed by stout silk or wire sutures passed through holes drilled on each side of the fracture sites, by the use of intramedullary wires or nails, or by lashing together oblique fractures. More popular and perhaps preferable, is the steadying of the floating segment by direct traction (Fig. 63). In severely ill patients this can be achieved simply under local anaesthesia by encircling one or more of the ribs in the unstable segment with towel clips, to each of which is attached a cord and a light weight (3–5 pounds) hanging over a pulley. Alternatively strong steel wire protected by polythene tubing can be placed round the ribs exposed through a small incision. Where possible the periosteum is elevated and the wire placed extrapleurally. Traction is usually required for 7–10 days.

These direct methods are frequently associated with the use of tracheotomy and can be applied to patients with bilateral injuries.



FIG 63—Rib traction. (From Proctor and London (1955) by courtesy of the authors and *British Journal of Surgery*)

Relief of anoxia

The treatment of oxygen lack in the tissues has recently become far more rational in chest injuries it is no longer permissible to think that oxygen therapy alone is sufficient. Anoxic, anaemic and stagnant anoxia present their own particular problems. Although classical indications for oxygen may be cyanosis, dyspnoea and a fast pulse it is important to remember that anoxia may exist without dyspnoea and that cyanosis is unreliable as a clinical guide and does not develop in patients with a haemoglobin content of 5 g. or less per 100 ml. There is a tendency to rely increasingly on accurate information provided by oxygen estimations on blood obtained by arterial puncture. The need for adequate blood transfusion in anaemic and stagnant anoxia is obvious. It is in the treatment of anoxic anoxia that confusion is steadily being dispersed. When this condition is due to mechanical causes such as obstructed air passages, compression of the lung by a pneumothorax, haemothorax or pyothorax or inadequate movements of the chest wall (flail chest or the stage of recovery from anaesthesia) their active treatment must precede or accompany oxygen therapy. In the past, all too frequently a patient with a crushed chest was left unaided in an oxygen tent while secretions accumulated in the air passages. Coughing was rendered ineffective by morphine, barbiturates, or a chest wall whose function was hopelessly impeded by a moving segment or because the underlying lungs were compressed by liquid or air in the pleura. Once the air passages are clear and the lungs can ventilate properly oxygen is of the greatest value. It should be used to prevent rather than cure anoxia, for example, by its use in the immediate post-operative phase. Oxygen tents are being used to a lesser extent in most instances oxygen can be given through a modern plastic oro-nasal mask, such as the Polymask.

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Oxygen at 8 l a minute will provide 60 per cent oxygen in the alveoli; this is adequate for most patients. Oxygen given for any period ought to be properly moistened; this is not a simple problem as bubbling a bottle of water is quite inadequate. Efficient humidifiers are now available and should be used.

Carbon-dioxide retention

Excess of carbon dioxide in the circulation does not cause cyanosis due to inadequate oxygenation of the haemoglobin. Recently the serious excess carbon dioxide has become widely recognized particularly in bronchitic and emphysematous patients whose pulmonary ventilation is poor, in whom a chest injury or operation carries high risks. In the early stages of carbon dioxide retention the stimulation of the respiratory centre and rise in arterial pressure may be deceptive. Carbon dioxide continues to accumulate and depresses the respiratory centre. Shallow breathing and failing consciousness occur in an oxygen tent without the gravity of the situation being appreciated. The colour and pulse may remain good. It is the steady lapse into coma that should be recognized for the need is then urgent to make sure that the lungs are properly ventilated. If active measures such as encouraging breathing, coughing fail the patient should be intubated, oxygen administered and carbon dioxide removed by the use of a Waters canister in the circuit. Tracheostomy is often the most efficient method of improving pulmonary ventilation. Such measures may likewise save elderly patients with injuries elsewhere than in the chest.

Thoracotomy in civilian chest injuries

Thoracotomy after injury is most likely to be required to stop bleeding which continues to be rarely employed. The low pressure in the pulmonary system allows collapse of the lung to be effectual in checking bleeding. Most bleeding in the pleural cavity comes from the chest wall and is treated by simple aspiration of the haemothorax, major surgery being invoked only for the case of clotted or infected haemothorax. In stab wounds or other penetrating injuries the heart and certain vessels may be injured. Clinical evidence of active bleeding warrants thoracotomy in suspected wounds of the heart and great vessels such as the superior vena cava, innominate and subclavian vein which have been successfully repaired. Even in the case of stab wounds of the heart itself conservative management has an important place. Probably less than 30 per cent of such wounds require exploration and in some of these the bleeding will stop to have stopped spontaneously. If cardiac tamponade (pallor, cyanosis, raised systemic blood pressure with poor peripheral pulses and cold limbs) continues despite effectual pericardial aspiration, the heart should be explored. Dr Cooley and Creech (1955) treated 57 patients with cardiac wounds, 28 conservatively and 14 by open surgery. The death rate was far higher in those treated surgically but this group included a higher proportion of gravely ill patients in the conservatively treated group.

If thoracotomy discloses bleeding from a large vessel it is well to remember that ligation of a pulmonary artery (even the main vessel) will not be followed by gangrene of the lung because the bronchial arteries provide an adequate

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tion. If however a large pulmonary vein is ligated the lung tissue which it is draining will die. Resection of such tissue usually by lobectomy is therefore required

Thoracotomy is required for a clotted infected haemothorax whatever the nature of the original injury. The operation is usually performed 3-5 weeks after the injury but should be done only after determined aspiration has failed. At thoracotomy the clot is removed and the visceral pleura relieved of its fibrinous envelope

Air embolism

Sudden deterioration and death in patients with chest injuries is sometimes due to air embolism complicating a lung injury. Death usually occurs within 2-3 days of injury but the embolism may occur after a week or longer. The condition is not an uncommon finding at necropsy of patients with severe chest injuries and was found in about 20-30 per cent of such cases by Sevvitt (1959). The embolism is on the left and not the right side of the heart which indicates entry through a pulmonary vein. A tear in the lung produced by a perforating fractured rib may be obvious at necropsy but in some cases a pulmonary lesion is difficult to find. The way in which air enters the circulation is not always clear. Air embolism is commonly associated with a tension pneumothorax but in some cases there is no evidence of pneumothorax then air probably enters through suction of alveolar air into a torn vein. The condition should be suspected when a patient suddenly collapses unexpectedly following apparent improvement. There is no specific preventive or curative treatment yet available

COMBINED INJURIES OF THE CHEST AND ABDOMEN

Whilst it is often possible to decide that a wound or closed injury has involved the chest alone or the abdomen alone it may equally be difficult to make any such clear distinction. Only the thin layer of the diaphragm separates the two cavities and any lesion affecting the diaphragm may encroach upon them both. Moreover clinical diagnosis is often confused by the fact that the abdominal parietes are supplied by the lower six thoracic nerves. Irritation of which in their thoracic course can lead to abdominal guarding, rigidity and even tenderness. Likewise irritation of the underside of the diaphragm by intestinal contents, blood or distension will embarrass respiratory function.

In many closed injuries of the trunk the anatomical separation of the two cavities is ignored by the mechanism of injury. Torison injuries produce splits which may involve both whilst some of the more violent blows to the whole chest which result from traffic accidents and falls from a height are liable to involve the juxta thoracic abdominal organs—the liver, spleen, kidneys and suprarenal glands, occasionally the oesophagus or stomach—as well as the diaphragm, chest wall, lungs and mediastinal structures. Fortunately widespread impact of this nature rarely produces lesions of the gut but even the relatively simple surgery for the arrest of bleeding from the liver or spleen may carry grave risks to life on account of the associated thoracic damage.

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Open wounds of the chest, whether perforating or penetrating, usually require surgical exploration. This is all the more essential if there is suspicion that the

diaphragm has been traversed by the wounding agent. The decision whether to operate through a thoracic or an abdominal approach remains a matter of dispute. Laparotomy is usually wiser for penetrating wounds (that is, those with a retained foreign body) which have passed from the chest into the abdomen. If however the surgeon has sufficiently exact evidence of the path of the wound to exclude injury to abdominal viscera other than the spleen, liver kidney upper half of stomach or splenic flexure of the colon a thoracic approach is wiser it can be extended if necessary to open the abdomen. The diaphragm is certainly more easily repaired from above, and damage to the above mentioned organs can often be dealt with through the chest the diaphragm being split and subsequently sutured. If laparotomy only is done, great attention must be paid in the post operative period to thoracic complications such as atelectasis and pleural infection. If anything chest complications are fewer after a thoracic incision possibly because they are anticipated and prevented or treated earlier perhaps because the patient has been operated on by a chest surgeon who would choose this route for preference. The clearing of bronchial passages and aspiration of pleural effusions remain vitally important manoeuvres.

CLOSED THORACO-ABDOMINAL INJURIES

Anatomical diagnosis is often extremely difficult, but it is possible to divide such injuries into three main categories from the point of view of their handling

Moderate chest injuries with a suspicion of an important abdominal lesion—If in fit young patients, a short period of observation has not resolved the diagnostic doubt, it is probably wiser to make an early decision to explore the abdomen. The chest condition is likely to become worse for a time, while the development of any degree of ileus following a lesion such as retroperitoneal haematoma will embarrass the chest and increase the risk of surgery should laparotomy be required after all. In elderly patients it is sometimes wise to gamble on the abdominal lesion being one that may not need surgery although with our growing understanding of the treatment of the thoracic component, especially by early tracheotomy it may well be that laparotomy is after all less risky than hopeful expectancy

Moderately severe chest and abdominal injuries—These require early and adequate transfusion, immediate attention to the chest lesion tracheotomy, chest aspiration and abdominal exploration as soon as the circulation and lungs appear to be functioning adequately

Severe chest and abdominal injuries—In the most severe injuries of this type a further weighty factor emerges. All the elements of a thoracic and juxta thoracic injury which can interfere with the function of the lungs as organs for gaseous exchange can also interfere with the pulmonary circulation (Fig. 64). The left heart cannot put out what the right heart does not deliver. Serious blood loss will often require replacement but transfusion may produce only temporary improvement, followed by venous congestion and pulmonary oedema. The exsanguinated patient is being transfused too fast in relation to the state of the pulmonary circulation. The only hope lies in doing everything possible to improve pulmonary function and at the same time carrying out transfusion. Both needs are equally urgent and one cannot wait on the other

CLOSED THORACO-ABDOMINAL INJURIES

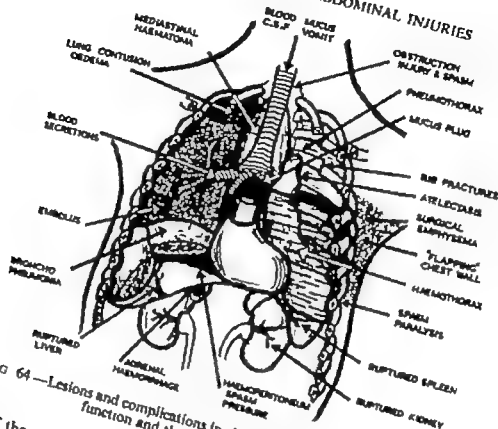


FIG 64—Lesions and complications in closed trunk injuries affecting lung function and the pulmonary circulation.

Many of those patients at present dying from trunk injuries following accidents on the roads have a chance of survival if they reach hospital within half an hour provided steps are taken immediately to restore blood volume and open up the lungs so that oxygenation is possible and the pulmonary vascular tree can allow a useful distribution of the much needed blood.

Cholethorax and chylothorax

Exceptionally cholethorax and chylothorax may follow compression injuries to the trunk. Both are detected by the aspiration of fluid from the pleural cavity. Bile in the pleural cavity almost invariably leads to infection. As soon as it is detected, closed water-seal intercostal drainage should be instituted. Chylothorax is a more serious problem. A chylous effusion may follow a relatively mild injury to the lower thoracic region usually either a direct blow or indirect injury associated with hyperextension of the spine. If the chylous effusion is removed completely and repeatedly by aspiration the lung will re-expand and in over one half of the patients spontaneous cure will follow. Continued outpouring of chyle with the loss of its water fats and proteins will lead to death by inanition if the daily loss is undiminished after 3-4 days, continuous closed drainage should be employed. If this fails surgery is indicated before the patient wastes. The thoracic duct is sought through a left lower thoracotomy. A collection of chyle will be found between the aorta and the oesophagus any area suggestive of the leaking duct should be sutured and the whole area generously packed with Gelfoam.

Adrenal apoplexy

Adrenal apoplexy is a relatively common complication of severe closed injuries to the chest or abdomen and when bilateral it is a potential source of acute adrenal failure (Sevitt, 1955). A blood clot occupies part or the whole of the medulla and the cortex is stretched around it; the gland is enlarged and distorted in its transverse axes and may be 2 cm. or more thick.

After chest and abdominal injuries the condition probably results from crushing of the gland against the rigid vertebral column. Sevitt (1955) found apoplexy in one or both glands in 25 per cent of 60 patients dying after closed thoracic, abdominal or pelvic injuries or multiple injuries involving the trunk. In most cases the injuries were so severe that death was not surprising in spite of prompt and determined treatment but in a small minority the clinico-pathological findings suggested that adrenal failure contributed to or accelerated death. This condition is another possible cause of irreversible shock (see Chapter 2) but only when both glands are affected so that adrenal failure occurs. Even though the cortex may be intact and secreting, its hormones cannot pass out through the compressed or disrupted central vein. Clinically adrenal apoplexy is very difficult to diagnose. Tests for adrenocortical hyperactivity such as blood eosinopenia and sodium retention by the kidney are probably the simplest to carry out. Cortisone therapy may be worthwhile in patients with thoraco-abdominal trauma who collapse unexpectedly but causes other than possible adrenal failure from apoplexy should be excluded.

REFERENCES

- d'Abreu, A. L. (1954) *A Practice of Thoracic Surgery*. London: Arnold.
- De Bakey, M. E., Cooley, D. A. and Creech, O. Jr (1955) "Aneurysm and Occlusive Diseases of the Aorta. Analysis of 203 Cases treated by Resection and Homograft Replacement." In *Cardio-Vascular Surgery Symposium*. Ford Foundation. Philadelphia: Saunders.
- Bahrson, H. T. (1955) "Surgical Treatment of Aneurysm." In *Cardio-Vascular Surgery Symposium*. Ford Foundation. Philadelphia: Saunders.
- Clarke, R. (1954) *Lancet* 2, 877.
- (1959) *Brit med J.*, 1, 125.
- Collier, H. S. (1954) *J. Kentucky med. Ass.* 52, 504.
- Proctor, H. and London, P. S. (1955) *Brit J Surg* 42, 622.
- Sevitt, S. (1955) *J. clin. Path.*, 8, 185.
- (1959) Personal communication.

CHAPTER 16

VENOUS THROMBOSIS AND PULMONARY EMBOLISM

S. SLVITT

THE DRAMA of sudden death from pulmonary embolism after injury, operation or childbirth is well known and pulmonary embolism has become increasingly recognized as a common cause of illness and death even in various medical conditions particularly after coronary thrombosis and in other heart disease. In accident surgery pulmonary embolism is quite common especially in middle aged and elderly people. It was found in 20 per cent of autopsies on injured patients in the Birmingham Accident Hospital (see Table). This is a much higher incidence than is generally thought and since it is a major problem it requires major consideration and action in any centre concerned with the treatment of accidents particularly as efficient anticoagulant drugs are now available for prophylaxis.

TABLE

FREQUENCY OF PULMONARY EMBOLISM AT AUTOPSY IN INJURED AND BURNED PATIENTS AT THE BIRMINGHAM ACCIDENT HOSPITAL

Embolism was the cause of death in 86 of the 103 patients affected

Main injury	Total cases	Number with pulmonary embolism	Percentage with pulmonary embolism
Fractured neck of femur	110	52	47
Other fractures of femur	17	9	53
Fractured tibia and fibula	9	5	55
Fractured pelvis (3 also with fractured ribs, 1 with head injury)	40	11	27
Abdominal and/or thoracic injuries	72	4	5.5
Head and thoracic injuries (9 also with abdominal and 5 with pelvic injuries)	46	1	2.2
Head injury	131	6	4.6
Fractured spine	21	3	14
Miscellaneous trauma	11	3	27
Total injured patients	457	94	20.5
Burns	158	9	5.6

DEEP VEIN THROMBOSIS

Embolism follows the mechanical accident of detachment of thrombi in the lower venous tree. Recent thrombi are attached to the walls of veins only at bifurcations and valve cusps and float almost freely in the blood stream. The thrombotic process is abacterial and histologically non-inflammatory so that the distinction between so-called thrombophlebitis and phlebothrombosis made on clinical grounds is not justified. Antibiotics have no effect on thrombophlebitic symptoms and histology.

shows that fresh thrombi are laid down on normal endothelium. Thrombosis is usually a silent process and only a minority of cases show clinical effects.

Sites of thrombosis

There have been two conflicting views as to the sites of origin of thrombi. The conclusion that thrombi always originate in the calf veins is based on the clinical finding that symptoms of venous thrombosis, particularly limb swelling and pain, commence in the calf. On the other hand the iliofemoral concept of primary thrombosis (Aschoff 1924) is based on vein dissections at autopsy on patients with clinical thrombosis. These conclusions have now been resolved by analysis of the results of vein dissections in cases reaching autopsy with or without clinical evidence of thrombosis (see Frykholm 1940, Gibbs 1957, also Sevt, unpublished observations).

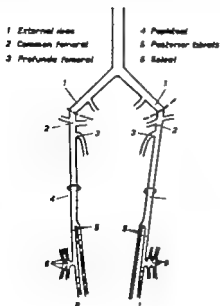


FIG. 65.—Diagrammatic outline of the lower venous tree showing the main primary sites of deep vein thrombosis. These are independent of each other although frequently thrombi are present in a number of them.

Our own autopsy studies of the sites and distribution of venous thrombosis have shown that there are six common but independent sites of primary thrombosis, four in the iliofemoral popliteal channel and main femoral tributaries and two in the leg veins (Fig. 65). These are (1) the iliac vein generally the external iliac just above the inguinal ligament, (2) the common femoral vein, including the mouths of the medial and lateral circumflex veins, (3) the termination of the deep femoral vein, (4) the popliteal vein, (5) the posterior tibial veins, and (6) the intramuscular veins of the calf particularly the soleal veins. This conclusion is derived from the various patterns of thrombosis which may be found (Fig. 66). Thrombi are often present in veins of the calf and absent in thigh veins and *vice versa* thigh vein thrombosis can be restricted to the common femoral vein in some limbs and to the deep femoral or popliteal veins in others. Thrombosis in the external iliac vein may or may not be associated with thrombosis in the common femoral vein whilst thrombosis in the soleal and posterior tibial veins can be associated or independent. Thrombi are most frequent in the deep calf veins (75 per cent of cases

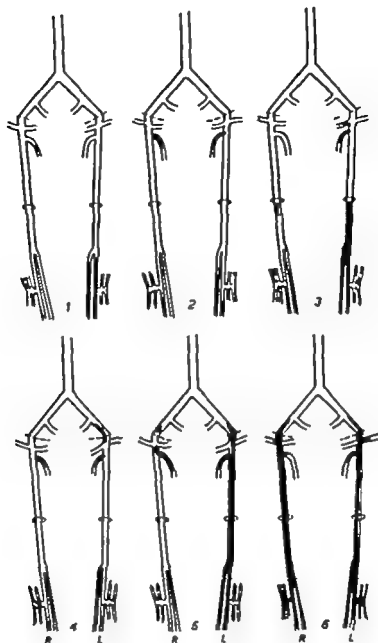


FIG. 66.—Diagrams of the sites of deep vein thrombosis found at autopsy in the lower limbs of injured patients. Cases 1-4 are selected to demonstrate the independence of thrombosis in the six main primary sites (see Fig. 65). Note particularly that thrombi in various thigh veins form independently of thrombi in leg veins and *vice versa*. Cases 5 and 6 show unilateral and bilateral continuous thrombosis from the iliofemoral to the posterior tibial veins. Thrombosis of the superficial femoral vein occurs after venous blockage at a higher level and rarely from upward propagation from thrombosis in the popliteal or posterior tibial veins. The patterns of thrombosis vary considerably in different patients. Moreover the pattern found in one limb is often different from that in the other.

with thrombosis) are common but not quite so frequent in the iliofemoral and main tributaries and are less common in the popliteal vein. Although thrombi separate from those in other veins can be found in the iliac femoral deep femoral medial and lateral circumflex popliteal posterior soleal veins, they are rare in the superficial femoral vein. When the latter bosed the thrombosis is continuous from the common femoral to the vein and often to the posterior tibial veins. This occurs in a minority of cases usually the result of obstructive thrombosis in the common femoral vein at a higher level and rarely of upward propagation of thrombus from the popliteal veins. Similarly a long continuous thrombus in the deep femoral vein is obstruction at its mouth.

Propagation of thrombi in the direction of the venous flow does occur generally limited in extent and its role has been exaggerated. Occasionally even it is extensive and thrombus may extend for example, from the femoral or iliac vein into the inferior vena cava.

Thrombosis is bilateral in about two-thirds of the cases with thrombi in both limbs but the pattern in one limb is often different from that in the other (Fig 1).

Frequency

Estimates of the incidence of venous thrombosis among medical and surgical patients range from 44 to 60 per cent (Gibbs, 1957. Hunter and his co-workers, 1945. Neumann, 1938). In our own series of autopsies on injured patients were found in 60 per cent of cases without evidence of pulmonary embolism. The total frequency would be even higher if cases with pulmonary embolism were included.

The clinical evidence depends on watchfulness and criteria of diagnosis. The incidence of clinical thrombosis is higher than is generally believed but it is a fraction of that seen at autopsy. Clinical thrombosis was reported in 12-15 per cent of patients with injuries to the legs (Bauer 1946. Aurin and Herrmann). In our own observations have shown clinical thrombosis in about 1 in 4 elderly patients with a fracture of the hip whilst autopsy dissections revealed thrombosis in 75 per cent of the cases. This evidence indicates that only about one-third of those patients with thrombosis who survived developed clinical signs of the disease because a similar clinical incidence was found in survivors and in those who died. Thus, thrombosis is a silent process in the majority of cases and this explains the numerous cases of pulmonary embolism unheralded by leg swelling or pain (see below).

Age survival period and bed rest

The frequency of thrombosis as found at autopsy increases with duration of bed rest and advancing age. The great majority (75 per cent) of injured patients over 60 years of age die with thrombosis in the lower venous tree; the frequency is progressively less in younger age groups but even in those under 15 years of age it is not unimportant (30 per cent of cases). The frequency increases rapidly during the first week after injury and thrombosis is found in 70-90 per cent of injured patients dying after this. The risk of thrombosis is particularly high in elderly patients who survive a week or longer and is less frequent in those under 50 years of age.

DEEP VEIN THROMBOSIS

important in choosing the groups of patients for routine prophylaxis with anti coagulant drugs (see below). Nevertheless the risk of thrombosis within 7 days or even within 3 days of injury is not negligible particularly in the elderly.

Nature of injury

Although it is often supposed that injury to a lower limb predisposes to thrombosis, analysis of the frequency in injured cases reaching autopsy does not support this. The nature of the injury does not directly affect the frequency of thrombosis but its influence is indirect. For example patients with injury to the head or head and chest are less likely (23 per cent of cases) to have venous thrombosis discovered by autopsy because many are young and most of those who die succumb within a few days of injury. On the other hand venous thrombosis is usually found at autopsy in 80-90 per cent of cases with a fractured neck of femur because most of them are elderly and survive more than a week. Studies in elderly medical patients who had been in bed a week or longer before death have also shown a high incidence of venous thrombosis at autopsy and this supports the conclusion that the location of injury or operation plays little direct role in thrombosis.

Side of injury and thrombosis

When clinical thrombosis occurs in a patient with an injury to one lower limb it is usually on the same side because of this injury is said to increase the likelihood of thrombosis in the affected limb. Our autopsy studies of a series of patients with a fractured femur or tibia showing venous thrombosis at autopsy do not support this contention. Thrombosis was generally bilateral (72 per cent of cases) and in the unilateral cases thrombosis was as common in the uninjured as in the injured limb. The undoubted clinical association of side of injury and thrombosis requires an explanation. Two other influences are at work. Sometimes the thrombosis is more extensive on the injured side but in other cases this explanation will not suffice and immobilization of the limb may be responsible. This, when combined with bed rest, probably reduces venous drainage and predisposes to swelling when venous thrombosis is present. It may be that the prolonged swelling of a fractured leg which is not infrequent is often the result of deep vein thrombosis. This may be at first confused with the residue of swelling from the acute injury or later hidden in a plaster cast.

Aetiology

Advancing age, lack of movement and duration of bed rest are above all the most important predisposing factors, each increasing the likelihood of venous stasis. The veins of the lower limbs depend for their emptying on muscle contraction: those of the elderly confined to bed will fill and the blood will tend to become stagnant. This is facilitated by the increase with age in tortuosity, diameter and number of the intramuscular veins, particularly those of the soleus muscle. The common denominator of venous thrombosis in obstetrical, surgical, traumatic and medical cases is venous stasis and bed rest plays the major role; the reason for the confinement to bed is less important.

The haematology of thrombosis cannot be considered here except to say that the fibrin thrombi extend from minute platelet foci deposited at valves and junctions.

the extension depends on an interplay of thrombogenic, antithrombogenic and fibrinolytic activity. Fibrinolysis is important in removing thrombi but its effect is limited by extensive thrombosis and venous obstruction. Organization from the wall of the vein may then occur. There is evidence that many thrombi disappear spontaneously and that this is accelerated in patients undergoing anticoagulant therapy. The latter antagonizes thrombogenesis but not fibrinolysis.

Anatomical factors

Anatomical factors decide where platelet foci are initially deposited, where the thrombi begin to form and the sites from which they extend.

The soleal veins are commonly involved because they become distended in the horizontal position in bed. Stagnant thrombosis is facilitated by their shape, course and size—they form a number of intramuscular arcades which increase in width and number with advancing age (Gibbs, 1957). Posterior tibial vein thrombosis is possibly encouraged by the relationship of the vein to the upper edge of the soleus muscle deep to which it crosses. Thrombosis in the popliteal vein is related to the adductor opening by which the vein may be compressed from the bulk of overlying muscle. Profunda femoral vein thrombosis commences at the termination of the vein or at the valve guarding the ostium because of the eddying of a stagnant flow. Thrombosis in the common femoral vein is facilitated by the inguinal ligament which may compress the vein and produce a pool of stagnant blood particularly when the thighs are flexed on the trunk as in Fowler's position. Eddying of the slowed flow at the valves and junctions of the medial and lateral circumflex veins and other tributaries initiates local thrombi. Eddying probably initiates thrombosis in the external iliac veins just above the inguinal ligament when the body is horizontal there is an acute weir-like change in direction of the slowed column of blood. Thrombosis is somewhat more common in the left iliac vein because the left common iliac vein is crossed and probably compressed by the origin of the right common iliac artery (Aschoff 1924).

Clinical diagnosis

The diagnosis of deep vein thrombosis need not be elaborated here except to say that pain or tightness are early symptoms but are often absent. That unilateral swelling of the limb particularly the calf is the most reliable sign and that pain on pressure in the calf or along the course of the vein or on dorsiflexion of the foot is commonly absent. Swelling is a sign of venous obstruction often of the femoral or popliteal veins, but is relatively uncommon because of the extensive venous anastomoses. Correlation of clinical with autopsy findings indicates that in about two-thirds of patients and about three-quarters of the limbs venous thrombosis is symptom-free even to the most careful observer. Bauer (1946) reported that phlebography was of great help in early diagnosis and suggested that it should be carried out whenever thrombosis or embolism is suspected. It too has its limitations because failure to demonstrate a vein may be due to technical reasons as well as thrombosis. Most important perhaps is the relative inability to demonstrate the iliac and common-femoral channels after the usual injection of contrast medium into a foot vein.

PULMONARY EMBOLISM

Detachment of thrombus is often associated with sudden movement effort or stress of which straining at stool is the classic example. Surgeons are not unfamiliar with collapse and death from embolism during manipulative reduction of a fractured femur or tibia. Fatal embolism sometimes follows active or passive movement of the lower limbs in bed transfer from bed to chair or *vice versa* turning of the patient from one side to another and ordinary ambulation. Detachment of thrombus and a sudden increase in venous flow both appear to be necessary for embolism to occur.

Analysis of the embolic episodes in 103 of our patients has shown that the frequency rises steeply during the first week, reaches its maximum during the second week and thereafter declines, slowly at first and then more quickly. A few episodes occur in the first 4 days whilst about half of them take place between 5 and 14 days after injury. 20 per cent occur in the third week and 10 per cent in the fourth week. After a month embolism is less frequent because the thrombi have lysed or are becoming organized to the venous walls. Fresh thrombi often a result of consequential bed rest are responsible for the 20 per cent of embolic attacks which occur during the second third and occasionally the fourth months after trauma.

Morbid anatomy

Anatomical classification of emboli is of some importance because the outcome depends to a large extent on the length, diameter and number of thrombi which become detached and carried to the heart and lungs. This is not to underestimate the reaction of the pulmonary arterial tree or of the heart to sudden blockage of a pulmonary vessel but this cannot be considered here.

Fatal emboli

Fatal emboli fall into two main groups, large wide-calibre emboli and multiple fine narrow emboli.

Large wide-calibre embolus—This is about 1–1.5 cm. in diameter and generally 10–30 cm. long, but the length varies considerably. Sometimes it is as short as 5 cm. or even as long as 40 cm. The long thrombi are often bent or coiled or broken up into a number of shorter emboli during their passage through the heart. They are often found straddling the bifurcation of the pulmonary artery or blocking one or both main arteries at the hila of the lungs. Sometimes the hilar vessels are empty and one, two or more primary intrapulmonary branches are blocked. In some cases a single shorter embolus is found blocking a main branch in one lung. About 70 per cent of fatal emboli fall into this group. Death is usually sudden but some patients die after gradual deterioration or acute congestive cardiac failure (see below). Infarction is unusual even in patients who survive hours or days. Some patients survive the massive embolism only to succumb later to cor pulmonale.

Multiple fine narrow thrombi—These are 2–4 mm. in diameter and generally each 1–3 cm. long. They are found within small branches of the pulmonary arteries in both lungs towards the periphery and in central branches. This shower of emboli is responsible for about 20 per cent of cases of fatal embolism. Sudden collapse and death may occur but usually death is preceded by sudden or gradual deterioration over days.

Non-fatal emboli

Non-fatal emboli are also of two kinds and evidence of previous episodes is not uncommonly found at autopsy (1) a short piece of a large-bore thrombus blocks a large intrapulmonary artery haemorrhagic infarction may or may not be found (2) a few fine-calibre thrombi are lodged centrally or peripherally within the lungs. Infarction is absent and the embolism is apparently silent and without clinical significance however the possibility of a relationship to subsequent pneumonic foci has been raised.

Secondary thrombosis

Secondary thrombosis is not uncommon particularly with large-calibre emboli. The thrombosis may extend distally from the embolus in the blocked artery like branches of a tree occasionally growth by central propagation of the embolus is seen. Thrombosis usually occurs when death is delayed then the volume of the pulmonary arterial tree cut off from the heart is increased and, in those patients balancing between life and death the likelihood of a fatal outcome is augmented. Prevention of secondary thrombosis is the basis of urgent intravenous heparin therapy. This should be given without hesitation even though it antagonizes or inhibits the action of plasma fibrinolysin (plasmin).

Origin of the emboli

Post mortem dissection indicates that the majority of fatal emboli come from thrombi in an iliac or femoral vein (Aschoff 1924 Gibbs, 1957 Leriche, 1946 Vance, 1934 and the author's unpublished observations) and not from the calf veins as is believed by many clinicians. This is notwithstanding the fact that thrombosis of the calf veins is more frequent. Large calibre and long total length indicate a large-vein origin whilst the appearance of an empty iliofemoral vein in one limb with iliofemoral thrombosis in the other argues in favour of the former as the origin. The iliofemoral thrombus may be separate, and unrelated to calf vein thrombi; sometimes the latter are absent. Multiple small emboli often come from the calf veins but sometimes when the popliteal or femoral veins are found thrombosed, small thigh vein thrombi must have been responsible. Rarely the embolus comes from a thrombosed inferior vena cava.

Contrary to the general belief that the emboli always originate from limbs showing clinical thrombosis and from injured limbs our data show that the iliofemoral veins of the injured and uninjured limbs are equally likely to be the source of fatal embolism whether or not there is clinical thrombosis in the injured limb.

Clinical syndromes

A clinical diagnosis of embolism has not been made in many instances where embolism is discovered at autopsy and therefore the clinical incidence underestimates the real frequency. Embolism is often unsuspected because the variety of clinical pictures is not generally appreciated. Clinicopathological correlation has evaluated a number of syndromes as follows:

Sudden collapse and death

Sudden collapse and death within minutes or one or two hours is the best known picture. Sometimes death is delayed for 24-48 hours after collapse and it is in these

PULMONARY EMBOLISM

cases that the diagnosis is often missed, this is particularly regrettable because death might be avoided by urgent heparin therapy to prevent secondary thrombosis. Sudden collapse occurs in the majority (about 70 per cent) of cases of fatal embolism. The clinical state comprises features of acute asphyxia or acute 'shock' or a variable mixture of their symptoms and signs. Acute pain in the chest is sometimes present and may lead to an erroneous diagnosis of coronary occlusion. In most cases (80 per cent) the syndrome results from large-calibre bifurcation or hilar emboli but in a minority (about 10 per cent) multiple thin emboli in major or peripheral branches of the pulmonary arteries are responsible.

Infarction

The classical symptoms of infarction—sudden onset of pain in the chest with dyspnoea, cough and haemoptysis followed by signs of consolidation and a pleural rub—are relatively uncommon and are rarely fatal. A partial syndrome of chest pain, dyspnoea and cyanosis lasting 1–3 days is more usual.

Pneumonic syndrome

A picture like pneumonia is more usual than the infarction syndrome. Dyspnoea, tachycardia, pyrexia and variable signs in the chest often suggest bronchopneumonia but the patient fails to respond to antibiotics and improves with anticoagulant therapy.

Abscess formation

Sometimes pneumonia develops around an infarcted area of lung and the latter may break down to form an abscess. Pyrexia, respiratory symptoms and general illness may be prolonged and the patient succumbs to pulmonary infection, septicaemia or a second embolic attack. Both antibiotic and anticoagulant therapy are required.

Gradual deterioration and death

The patient gradually deteriorates and death finally occurs from bronchopneumonia or cardiac failure. Embolism is rarely diagnosed. This picture is not uncommon in elderly patients and had occurred in 20–25 per cent of our cases in whom a fatal embolism was found at autopsy. Large-calibre emboli in large or main pulmonary arteries were found in about one half and multiple small emboli in the remainder.

Acute congestive cardiac failure

Acute congestive cardiac failure occasionally dominates the picture and death occurs a week or two later. Blockage of one or more large branches is usually responsible. The possibility of pulmonary embolism should always be considered in patients developing congestive failure while confined to bed or after manipulative reduction of a fractured femur or tibia or other suspicious circumstances because urgent and prolonged anticoagulant therapy (heparin followed by phenindione) is needed. Death due to thrombotic extension of the arterial obstruction may then be avoided.

Chronic pulmonary hypertension and cor bovinum have recently become recognized as sequelae of pulmonary embolism. The pulmonary arteries become thickened and narrowed through fibrous inclusion of the emboli.

Acute hypotension possible with renal failure

In some cases an episode of hypotension lasting 12-24 hours or longer occurs for no apparent reason and coronary thrombosis may be diagnosed. Renal function may be affected and the blood urea may rise rapidly to 200 mg per 100 ml or more; this may or may not be associated with oliguria (see Chapter 7). Autopsy reveals one or more major emboli within major branches of the pulmonary arteries often with large areas of infarction; sometimes this has become infected and broken down to abscesses. The possibility of pulmonary embolism in cases of unexplained hypotension or renal failure developing days or weeks after confinement to bed should be considered because anticoagulant therapy is urgently needed.

Frequency of embolism

Pulmonary embolism is important in the deaths of many injured patients as well as many medical and surgical subjects. It was found in 20 per cent of autopsies on injured patients in the Birmingham Accident Hospital and in 15 per cent it was the cause of death (see Table). This corresponds to 0.7 per cent of patients admitted for more than 24-48 hours. The true frequency is higher because the figures exclude cases not reaching autopsy and clinical cases among survivors. The total incidence is certainly more than 2 per cent and the fatal incidence about 1 per cent of the patients admitted.

The literature concerned with the frequency of pulmonary embolism after injury is limited but also indicates that the risk is high. It must be noted that the autopsy frequency depends on the vigilance and technique of the observer. McCartney (1934) found embolism in 3.8 per cent of 1 647 autopsies performed on patients with miscellaneous trauma whilst Hamilton and Angevine (1946) found it in 6 per cent of 1 065 autopsies on battle casualties, many with injuries in the lower limbs.

The frequency among medical and surgical autopsies is also high: the figures range from 2.6 per cent (McCartney, 1934), 6 per cent (Cohn and Walsh, 1946), 9-14 per cent (Belt, 1934; Gibbs, 1957; Hampton and Castleman, 1940), 17 per cent (Hunter and his colleagues, 1945) to 23 per cent in a chronic sickness institution (Moran, 1947).

It is clear that pulmonary embolism is a common phenomenon and cause of death in all kinds of patients, medical, surgical and traumatic.

The frequencies based on clinical criteria are underestimates and are not comparable to those based on autopsy figures. In non-traumatic—that is medical, surgical and obstetric—patients, the published figures vary considerably, ranging from 1 in 150 to 1 in 3 000 cases of fatal embolism among the patients at risk.

Bauer's (1946) summary of recorded fatal embolism was 0.26 per cent after surgical operations, 0.4 per cent in medical cases and 0.033 per cent after parturition. Other figures, mainly from large series of surgical cases, are 0.085-0.14 per cent (Kistner and Smith, 1954), 0.19 per cent (Jorpes, 1947), 0.24 per cent (Wise, Loker and Brambel, 1949) and 0.7 per cent (Barker and his colleagues, 1945), (see also Zilliacus, 1946; Gjores, 1956).

PULMONARY EMBOLISM

Nature of injury and patient's age

The total incidence of pulmonary embolism in a series of injured patients reaching autopsy is always relatively high but it will vary in different series because of different proportions of different kinds of patients. This is because some patients are more likely to develop venous thrombosis and hence embolism than others who are more likely to die with little or no venous thrombosis and have consequently a lesser risk of embolism (see above). The difference is shown in the Table. Embolism was found in 47-55 per cent of autopsies on patients with a fractured femur or tibia and was the commonest single cause of death among them. These rates are even higher than McCartney's (1934) report of embolism in 15 per cent of autopsies on patients with a fractured tibia and in 25 per cent of those with a fractured femur but compare with that of Golodner, Morse and Angrist (1945) who found 9 cases of embolism among 25 autopsies on elderly patients with fractured hips. Patients with fractured hips are at particular risk. Indeed 10 per cent of a specially studied series of elderly patients admitted with fractured hips died of pulmonary embolism within 3 months (Gallagher and Sevlitt 1957-59).

Pulmonary embolism was also frequent among patients dying after a fractured pelvis (27 per cent) but it was not so common in those dying after abdominal thoracic or cerebral trauma (2-6 per cent) or after burning (5-6 per cent) (see Table). The different rates partly reflect differences in the frequency of deep vein thrombosis which in turn mainly depend on duration of bed rest and advancing age (see above). Those patients who die after fractures of the femur, tibia or pelvis are mostly elderly patients who survive and remain in bed long enough for extensive venous thrombosis to develop whilst many of those who succumb to head injuries, burns and other trauma are young people who die within a few days when venous thrombosis is unusual or limited.

Thrombus detachment rate

The thrombus detachment rate is an index of the rate and risk of embolism from thrombi in deep veins and is based on the frequencies of pulmonary embolism and deep vein thrombosis in a series of autopsies. There is some evidence that the risk of detachment varies in different groups of patients. The rate is very high in those with fractured hips (iliofemoral detachment rate about 50 per cent) and is significantly lower among burned patients and those with head or head and chest injuries (iliofemoral detachment rate 20-30 per cent). Longer survival periods which prolong the risk of embolism contribute to this difference and possibly also a greater extent of iliofemoral and other vein thrombosis. Gibbs (1957) reported a greater thrombus detachment rate among post-operative cases compared to others and thought that differences in mobility of the patients contributed to this.

Embolism in patients with clinical venous thrombosis

The risk of embolism is considerable when clinical thrombosis is evident although the embolus often originates from the other limb. This, of course, means that anti-coagulant therapy is imperative whenever a diagnosis of thrombosis is made. From an analysis of the literature Zilliacus (1946) and Bauer (1946) concluded that fatal pulmonary embolism occurred in 15-20 per cent of patients with diagnosed thrombosis following surgical operation or complicating medical diseases and in 3-5 per

VENOUS THROMBOSIS AND PULMONARY EMBOLISM

cent of cases following parturition. The true incidence is higher because embolism is often diagnosed only at autopsy. Pulmonary embolism occurred in 30 per cent of our patients with untreated clinical thrombosis following a fractured hip. The risk was about 3 times greater than in similar cases without clinical thrombosis, the great majority of whom probably had silent thrombosis—about 10 per cent of them developed embolism. Most emboli nevertheless come from clinically silent limbs and about half the cases are not preceded by any clinical evidence of thrombosis.

TREATMENT

Venous thrombosis

Anticoagulant therapy is aimed at the prevention of further thrombosis and embolism. When carried out efficiently it reduces the high risk of pulmonary embolism by 80–95 per cent; it also reduces the morbidity of persistent leg swelling and pain although this depends on how early treatment is begun (Ball and Hughes, 1946; Bauer, 1946; Marks, Truscott and Withycombe, 1954). Therapy by antiprothrombin drugs like ethyl biscoumacetate and phenindione is now well established. These drugs are effective and suitable and have replaced dicoumarol treatment, which was difficult to control. By 24–36 hours the prothrombin activity of the plasma is reduced to a level at which further thrombosis is unlikely. Dosage is affected by salicylates and by tetracycline and other antibiotics the use of which should be avoided if possible. Elderly patients need only one-quarter to one-half the maintenance dose for younger adults. Therapy is controlled by estimations of the 'prothrombin activity' of the plasma, daily for the first 3 days and then 2 or 3 times a week to steer a course between ineffective and hazardous (risk of bleeding) dosage. Dosage is aimed at prolonging the prothrombin time to between $2\frac{1}{2}$ and 3 times normal; this is equivalent to maintaining a 'prothrombin activity' level between 10 and 30 per cent of normal. Some workers (Marks, Truscott and Withycombe, 1954) advocate heparin for 24 hours as well as the antiprothrombin drug because it seems to reduce the duration of leg pain and the average stay in bed.

It is now customary to encourage active exercises in bed as soon as pain is relieved and to allow the patient up usually within a week of commencing therapy when the leg is no longer tender provided other conditions permit. Anticoagulant therapy is continued while the patient is in bed and for a week or longer after that. There is no doubt that the combination of anticoagulant therapy and early ambulation has been followed by a great improvement in mortality and morbidity although theoretically the limbs should be kept still for at least 2–3 weeks until the residual thrombi are becoming fibrosed to the veins. The ability to permit early movement and ambulation without appreciable danger is difficult to explain. It may be related to the unopposed and possibly increased fibrinolytic activity of the plasma of patients undergoing anticoagulant therapy. Thrombogenesis in the blood is inhibited but not fibrinolysis so that thrombi which are non-obstructive might disappear: their surface is continuously exposed to a column of flowing blood with fibrinolytic activity. This possibility requires investigation.

Pulmonary embolism

Intravenous heparin therapy is urgently indicated as soon as the diagnosis is suspected and should be given either intermittently or preferably by continuous

TREATMENT

drip for 24–48 hours. Dosage should be sufficient to double or treble the whole blood clotting time. Phenindione or other suitable antiprothrombin drug should be simultaneously given. heparin may be discontinued when the plasma prothrombin level has been adequately reduced.

The heroic Trendelenburg operation is rarely performed and very few successful cases have been reported. With adequate blood transfusion, subsequent antibiotic therapy and modern techniques it should probably be carried out more frequently in suitable cases. The surgeon is often reluctant to open the chest and pulmonary artery because he cannot foresee whether sudden collapse from embolism will result in death or spontaneous recovery. Speed is essential and perhaps the most suitable opportunity is when collapse during an operation or manipulation is suspected to be due to pulmonary embolism. In these circumstances the personnel and instruments required are likely to be immediately available.

Vein ligation

The enthusiasm of a few years ago for femoral vein ligation in certain centres in the United States of America has abated and now that the dust has settled this is not difficult to understand. Superficial femoral vein ligation has a protective value against pulmonary embolism whilst the price paid, a relatively high incidence of undesirable local sequelae, particularly chronic limb swelling, is high (Lillie Buxton and Duff, 1949; Szilagyi and Alsop, 1949). Protection is incomplete because unilateral ligation cannot prevent detachment of a silent thrombus from the other limb whilst bilateral ligation below the deep femoral veins cannot protect against detachment of a thrombus from the iliac or common femoral veins. Even if all clinical cases of thrombosis were fully protected, many instances of pulmonary embolism unheralded by clinical thrombosis would still occur. Although anticoagulant therapy is the treatment of choice, vein ligation may be necessary in certain circumstances. Bilateral ligation should be considered when drug therapy is contra-indicated, for example, in patients with peptic ulcer or in the rare event of failure to prevent pulmonary embolism, particularly when the attacks are repeated. Caval ligation might then be necessary.

Prophylactic vein ligation

Prophylactic vein ligation has been advocated in patients susceptible to venous thrombosis because of age, injury or disease (Allen, Linton and Donaldson, 1947). These authors ligated both superficial femoral veins of elderly patients with fractured hips and no case of fatal embolism occurred. The advent of safe, orally administered anticoagulant drugs has made prophylactic vein ligation almost completely unnecessary.

PROPHYLACTIC TREATMENT

Measures intended to prevent venous thrombosis and hence pulmonary embolism are exercise, movements and early ambulation to reduce venous stasis and the administration of anticoagulant drugs.

Exercise, movement and early ambulation

Exercises and movement in bed are necessary especially in middle-aged and

elderly people, to prevent inertia and immobility after injury or operation. Foot movements should be encouraged to exercise the important soleus muscle not merely wiggling of the toes, which has little effect. Certain practices like taut sheets and instructions to the patient to be still should be forbidden. Fowler's position should be prohibited at least for prolonged periods because it aggravates venous stasis below the inguinal ligament and in the dependent soleus muscle. Pillows under the knees are said to be harmful because pressure and knee flexion can reduce the flow through the popliteal veins. There is no easy rule to the prevention of venous stasis by exercises and movement. Mobility in bed frequent (half hourly at least) changes in position, passive and active exercises all play a part.

The problems are accentuated in the field of trauma, particularly in patients necessarily confined to bed, and are further aggravated through restrictions imposed by immobilization of a lower limb in a splint or plaster cast and in patients of advancing age perhaps with chronic diseases like congestive heart failure hypertension emphysema or senility. Early operation and early mobility are particularly indicated in elderly injured patients but unfortunately are often difficult or impossible to carry out. Nevertheless certain operations like pinning or nailing of fractured hips should be done almost as emergency procedures and within 24 hours of the injury so that the patients may quickly begin active and passive exercises and ambulation. Ambulation, of course, means walking and not merely early lifting out of bed on to a chair with the patient's legs hanging down but immobile.

Early post-operative ambulation has had some success but its advantages are limited by the not infrequent onset of venous thrombosis within a few days of confinement to bed. Thrombosis may also have already developed through bed rest before admission to hospital. If thrombosis has not occurred, ambulation may prevent it but if thrombosis is present sudden activity may produce embolism. The clinician is in a dilemma because most cases of thrombosis are silent, particularly at this stage and those patients at risk of embolism cannot be distinguished. Hunter and his colleagues (1945) found thrombosis at autopsy in 18 per cent of patients given exercises against venous stasis compared with 53 per cent among concurrent cases not exercised and non-ambulatory. Nevertheless many surgeons have been disappointed in early post-operative rising and prophylactic exercises (Blodgett and Beatue 1946 Powers, 1949 McCann, 1950) which did not appear to influence the annual rate of clinical thrombosis or embolism.

Anticoagulant drugs

Although anticoagulant therapy greatly reduces the incidence of pulmonary embolism among patients with clinical thrombosis the large number of embolic attacks in patients with silent thrombosis will still occur. Marks Truscott and Withycombe (1954) treated over 1 000 cases of venous thrombosis with a mortality from embolism of only 0.2-0.5 per cent but found that the annual incidence of fatal embolism in the hospital was unaffected. Their experience may be unusual but probably more than 50 per cent of cases of fatal embolism are unheralded by leg pain or swelling.

Prophylaxis with anticoagulant drugs offers the best chance of greatly reducing the incidence of venous thrombosis and pulmonary embolism. Prophylaxis means

PROPHYLACTIC TREATMENT

the use of a drug in a large number of patients at risk with the aim of preventing venous thrombosis and hence pulmonary embolism in all of them. Prophylactic heparin was found of great value (Bauer 1941 1946 Crafoord, 1941 Jorpes 1941 Murray and Best 1938 Wetterdall 1941) but enthusiasm was diminished by the expense of the drug. Later dicoumarol became available and was used with or without a preliminary course of heparin (Baker and his colleagues 1950 Barker and his colleagues 1945 Bruzelius 1945 Kistner and Smith 1954 Lehmann 1943 McCann 1950 Murray 1947 Wise Loker and Brambel 1949). These reports were not specifically concerned with injured patients and were generally related to the prevention of thrombo-embolic incidents after major surgery or in medical cases.

Unfortunately none can be considered a controlled trial all were essentially clinical in approach noting the incidence of diagnosed venous thrombosis and pulmonary embolism in the series. Usually but with a few exceptions the incidence was compared with the results of previous years and not with that of a concurrent untreated series. The patients varied widely in their ages clinical diagnosis, activity and no doubt in their liability to thrombosis. Mortality rates were not reported and pathological studies on the incidence of venous thrombosis and pulmonary embolism at autopsy were not made. Notwithstanding these criticisms taken together the reports indicate a definite measure of protection by the prophylactic use of anticoagulants. For example Wise Loker and Brambel (1949) compared the results of dicoumarol as a prophylactic measure after operation in 3 304 cases of major abdominal surgery with 2,030 concurrent cases not so treated. Thrombo-embolism was found in 1.6 per cent in the control series and in 0.18 per cent of the treated series. Embolic phenomena occurred in 0.4 per cent and 0.3 per cent and fatal embolism in 0.2 per cent and 0.03 per cent respectively. Kistner and Smith (1954) compared the results in about 12,000 patients undergoing major surgery half of whom were given dicoumarol. The incidence of non fatal embolism was 0.2 per cent and 0.12 per cent and of fatal embolism 0.085 per cent and 0.045 per cent in the control and trial groups respectively. Many of the failures resulted from too late too little or too short administration of the drug from difficulties inherent in its slow acting, irregular and relatively prolonged effects or to absence or difficulties of laboratory control. With the advent of newer antiproteolytic drugs like ethyl biscoumacetate and phenylindanedione (phenindione) which are quicker acting and non-cumulative laboratory control of dosage is easier and effective anticoagulant activity can be maintained for long periods without danger of significant haemorrhage.

A controlled clinical experiment using phenindione was begun in 1957 in the Birmingham Accident Hospital (Gallagher and Sevti, 1957-59). The trial has been restricted to patients over 55 years of age admitted with a subcapital or intertrochanteric fracture of the femur. This group of patients forms a relatively homogeneous clinical series with a particularly high incidence of venous thrombosis and pulmonary embolism (see above). Patients admitted on even days of the month were given phenindione from admission or from the day after admission—initial dose 150-200 mg. Pinning or nailing of the fracture was often carried out at operative or post-operative bleeding. The control series consisted of similar patients admitted on odd days of the month. Drug dosage was controlled by plasma

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prothrombin tests daily for the first 3 days and then 2-3 times a week and, in these elderly patients a relatively low dosage of 25-75 mg daily was usually sufficient to maintain an effectively low prothrombin activity of the plasma. Phenindione was continued during the period of bed rest and afterwards until the patient was making satisfactory progress on crutches: the period varied in different patients but was often 4-6 weeks occasionally 8-12 weeks. When necessary drug therapy was continued after discharge under domiciliary control of plasma prothrombin activity. The patients were followed up for a period of 3-4 months. Most of those who died in hospital or at home were submitted to necropsy and the pulmonary arteries and the major veins of the thighs and legs were fully dissected. This part of the investigation is novel and is considered particularly important.

The results are very encouraging. No case of embolism occurred among 150 patients under the influence of phenindione compared with an incidence of 9.7 per cent of fatal embolism and 13.6 per cent of total embolism among the 154 patients in the control series. There were 45 deaths (29 per cent) in the control series and 25 (16.6 per cent) in the trial series. The two cases of fatal embolism and the one of incidental embolism in the phenindione group occurred after therapy was stopped: this raised the difficult problem of when to stop prophylaxis in elderly inactive patients. Clinical venous thrombosis occurred in 27 per cent of the control series and in 2.7 per cent of the trial series. Significant thrombosis at autopsy was found in 30 out of 38 control cases studied but only in 3 of the 21 in the phenindione series examined—the ones with pulmonary embolism found at autopsy who had died after the cessation of prophylaxis. In most cases the whole lower venous tree was completely free of thrombi. Our evidence indicates that phenindione effectively prevents thrombosis in veins and eliminates the risk of pulmonary embolism in patients under its influence. Further work is necessary to define the indications and contra-indications to prophylaxis and to strike the balance between the indiscriminate administration of phenindione to every patient admitted with an injury and its restriction to narrow selected groups. Prophylactic protection should be given to those particularly at risk: that is those over 50 years of age about to undergo a period of bed rest for longer than 2-3 days and in whom anticoagulant therapy is not otherwise contra-indicated. This would include all middle-aged and elderly patients with a fractured femur or tibia and possibly most of those with a fractured pelvis. This policy should reduce the incidence of fatal embolism in the Birmingham Accident Hospital by 80-90 per cent. The drug should be continued until at least 1 week after the patient is reasonably mobile and ambulant. Therapy should be controlled centrally by the hospital laboratory by repeated prothrombin estimations using Quick's or another suitable method. This means the setting up of an organized and properly staffed anticoagulant unit within the laboratory. Major contra-indications are a history of recent haematemesis or peptic ulceration and the presence of a haemorrhagic diathesis. Chronic renal or hepatic disease are not considered contra-indications but less drug, more care and more frequent prothrombin estimations are needed. Cerebral contusion or recent injury to the spine are probably contra-indications because of the special dangers of a small amount of haemorrhage. These and other problems such as the risk of or freedom from excessive haemorrhage during and after different kinds of operations have to be worked out through further experience.

REFERENCES

REFERENCES

- Allen, A. W. Linton, R. R. and Donaldson G (1947). *J Amer med Ass.*, 133 1269
- Aschoff L. (1924). *Lectures on Pathology* New York Hoeber
- Aurn, F. B. and Hermann, L. G (1948). *Amer J Surg.*, 116 586
- Baker D V Warren R., Homans, J. and Littman, D (1950). *New Engl J Med* 242, 923
- Ball, A. P., and Hughes, H. O (1946). *Brit med J.*, 1 560
- Barker N. W., Cromer H. E. Hurn, M. and Waugh, J. M (1945). *Surgery* 17 207
- Bauer G (1941). *Acta med scand.*, 107 136
- (1946). *Lancet* 1 447
- Belt, T. H (1934). *Amer J Path.* 10 129
- Blodgett J. B., and Beattie, C. J (1946). *Surg Gynec Obstet.*, 82, 485
- Bruzellus, S (1945). *Acta chir scand* Suppl 100 92
- Crafoord, C (1941). *Acta med scand* 107 116.
- Cohn, R., and Walsh, J (1946). *Stanf med Bull.*, 4 97
- Frykholm, R (1940). *Surg Gynec Obstet* 71 307
- Gallagher N. G., and Scullis S (1957-59) To be published
- Gibbs, N. M (1957). *Brit J Surg* 45 209
- Gjores, J. E. (1956). *Acta chir scand.*, Suppl. 206 1
- Golodner H. Morse J. and Angrist, A (1945). *Surgery* 18, 418
- Hamilton, T. R., and Angeline D. M (1946). *Wills Surg.*, 99 450.
- Hampton, A. O., and Castleman, B (1940). *Amer J Roentgenol.*, 43, 305
- Hunter W. E., Krygier J. J. Kennedy W. J. C., and Sneed, V. D (1945). *Surgery* 17 178
- Jorpes, E. (1941). *Acta med scand.*, 107 107
- (1947). *Surg Gynec Obstet.*, 84 677
- Kistner R. W., and Smith, G. V (1954). *Surg Gynec Obstet* 98 437
- Lehmann, J (1943). *Lancet* 1 611
- Leriche, R (1946). *Lyon. chir.*, 41 143
- Lillie R. H., Baxton, R. W., and Duff I. F (1949). *Arch. Surg., Chicago* 59 609
- McCaun, J. C. (1950). *New Engl J Med.*, 242, 203
- McCartney J. S (1934). *Amer J Path.*, 10 709
- Marls, J., Truscott, B. M., and Withycombe J. F. R. (1954). *Lancet* 1 787
- Moran, T. J (1947). *Amer J clin Path.*, 17 205
- Murray D. G., and Best C. H (1938). *Ann Surg.*, 108, 165
- Murray G (1947). *Surg Gynec Obstet.*, 84, 665
- Neumann, R (1938). *Virchows Arch.*, 301 708.
- Powers J. H (1949). *Arch Surg., Chicago* 59 601
- Szilagyi, D. E. and Alsop J. F (1949). *Arch Surg., Chicago* 59 633
- Vance, B. M (1934). *Amer J Surg.*, 26 19
- Wetterdall, P (1941). *Acta med scand.*, 107 123
- Wise, W. D., Loker F. F., and Brambel, C. E. (1949). *Surg Gynec Obstet.*, 88 486
- Zilliacus, H. (1946). *Acta med scand* Suppl. 171 13

CHAPTER 17

MENINGITIS AFTER HEAD INJURY

S SEVITT

This chapter is concerned with meningitis after an apparently closed head injury in the absence of an obvious compound fracture or penetrating wound. A fracture is compound however when it involves one or more of the paranasal sinuses, the cribriform plate of the ethmoid bone or less commonly the petrous temporal bone and the middle ear and permits penetration of the nasopharyngeal bacterial flora. Most cases are pneumococcal and are amenable to chemotherapy so that it is important to recognize the complication early and treat it vigorously the price of failure is usually death. Before the antibiotic era few patients survived and those who did generally had serious sequelae like blindness, deafness or mental retardation. Now with early diagnosis and energetic therapy the majority survive and few have residual symptoms.

The condition is not common but this only partly explains the curious defect in medical teaching which allows many experienced practitioners as well as newly qualified doctors to be unaware of the hazard. Meningitis should be considered as a possible cause for unusual symptoms or unexpected illness in a patient with a recent head injury especially to the frontal area or the face otherwise the diagnosis will be made late or at autopsy. This is particularly unfortunate as the vast majority of cases follow minor concussion in which the cerebral prognosis is otherwise most favourable.

AETIOLOGY AND BACTERIOLOGY

The essential feature allowing meningitis to develop is a direct or indirect communication between the subarachnoid space and the nasopharynx. Most patients sustain a blow to the forehead or face and the anterior cranial fossa is fractured (Fig. 67a) part of the fracture extends into the cribriform plate of the ethmoid into the ethmoidal air sinuses or into a frontal sinus. In other instances there is a fracture of the middle cranial fossa involving the petrous temporal bone and the middle ear. The mucous membrane is torn and the wound permits intracranial invasion by the normal nasopharyngeal bacterial flora. Involvement of sinuses and risk of meningitis may also occur after injuries to the face (see Chapter 19) not involving the brain and cranium proper.

Escape of cerebrospinal fluid from the nose or ears indicates that the dura mater is torn but a tear may be present without escape of fluid. Autopsy often reveals a macroscopic dural tear over the fracture which allows the nasopharynx to communicate with the subarachnoid space via the lacerated arachnoid mater on the overlying injured surface of the frontal or temporal lobe. A torn dura offers a ready explanation for bacterial invasion but sometimes no rupture can be seen at

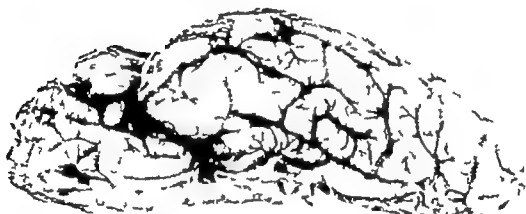


FIG 67—Fatal case of pneumococcal meningitis after a head injury (a) Fracture of the right anterior cranial fossa involving the cribriform plate of the ethmoid and the crista galli. This was associated with a dural tear and allowed pneumococcal invasion from the nasopharynx. (b) A small area of subarachnoid bruising on the orbital surface of the right frontal lobe related to the skull fracture here presumably bacterial entry occurred. The meningeal inflammatory exudate is thin and does not photograph well but is visible over the pons and the interpeduncular fossa.





(a)



(b)

FIG 68 —(a) Convexity and (b) undersurface of the left cerebral hemisphere of a patient with pneumococcal meningitis. Meningitis developed 14 days after a fracture of the ethmoid (with no concussion) and a course of intrathecal penicillin therapy supplemented by sulphadiazine and parenteral penicillin was instituted. The lumbar cerebrospinal fluid became sterile and its cell count and protein concentration fell quickly but the patient remained unconscious. Three days later right-sided twitching commenced and he died 5 days after the beginning of treatment.

Note that there is no evidence of meningitis on the undersurface of the hemisphere but the upper two-thirds of the convexity is covered with a thickened greyish meningeal exudate in which pneumococci were demonstrated. There was no evidence of active meningitis over the other hemisphere. Death was presumably due to a persistent unilateral meningitis and diffuse subarachnoid blockage the latter preventing circulation of penicillin-containing cerebrospinal fluid. Hydrocephalus was absent in this case because the blockage affected only one hemisphere.

PROPHYLAXIS

autopsy. Bacterial invasion must then have occurred through a damaged but continuous sheet of dura. Repeated attacks may occur when the dura is torn.

Most patients have little clinical or pathological evidence of brain damage (see Fig. 67b) conversely meningitis rarely develops if there is severe cerebral trauma possibly because swollen locally damaged cortex plugs the dural tear. In some instances the onset of meningitis days or weeks after injury may result from diminution of the local brain swelling and opening of the tear.

Most infections are with pneumococci (*Streptococcus pneumoniae*) a minority involve other streptococci or are abacterial (see below). Strangely no cases of infection with *Haemophilus influenzae* or Friedländer's bacillus have been reported even though these organisms are also common in the nasopharynx. In the author's series of 25 cases 17 were pneumococcal 3 were infected with non haemolytic streptococci (1 a group D faecal streptococcus) 1 was meningococcal and 4 were abacterial. The pneumococci found are the usual variety of serological types which normally inhabit the nasopharynx. Fortunately almost all of them are sensitive *in vitro* to penicillin chloromycetin sulphonamides tetracycline and other antibiotics. Penicillin resistant strains of pneumococci if they occur are very rare but one of the 35 cases reported by McKendrick (1954) was said to be infected with a penicillin resistant pneumococcus.

Unlike the meningococcal form pneumococcal meningitis rarely occurs *de novo*. Usually it is a complication of infection with pneumococci elsewhere such as otitis media sinusitis mastoiditis or pneumonia but a significant minority have a recent or previous history of head injury and skull fractures are often demonstrated. Thus head injury preceded meningitis in 5 of the 38 cases of Smith, Duthie and Cairns (1946) in 7 of the 125 cases of Appelbaum, Nelson and Albin (1949) in 4 of the 27 cases reported by Galloway and Chambers (1953) in 5 of the 102 cases treated by Alexander, Flippin and Eisenberg (1953) and 3 of the 35 cases described by McKendrick (1954) had skull defects. Head injury can thus account for about 7 per cent of cases of pneumococcal meningitis. Nevertheless meningitis, from any cause after civilian head injuries is infrequent and only 2-3 cases are seen every year at the Birmingham Accident Hospital among about a thousand patients with head injuries of all degrees of severity.

PROPHYLAXIS

Chemoprophylaxis is indicated whenever a fracture of the face or cranium is known or suspected to communicate with the nasopharynx, an air sinus or the middle ear. This includes all patients with cerebrospinal rhinorrhoea and otorrhoea and those bleeding from the ear nose or mouth in whom local trauma cannot account for the haemorrhage. Of course therapy should also be given to patients with compound fracture of the skull and may be extended to include all those with any skull fracture. Unfortunately radiological diagnosis of fractures into *sinuses* is difficult and apparently negative findings are often inconclusive.

Definite indications should be laid down for junior medical staff in hospitals because chemoprophylaxis and close observation require a stay in hospital for 7-10 days or longer and most of the cases of meningitis occur among patients with relatively slight cerebral injury who otherwise might be discharged within

24-48 hours of admission Too wide a net would detain far too many patients too long in hospital whilst too small a net might allow cases to slip through and develop meningitis The major indication for chemoprophylaxis is a fracture of the anterior cranial fossa and fracture should be presumed when there is a large orbital haematoma indicated by an extravasation of blood into the eyelids or a diffuse subconjunctival haemorrhage after a face or forehead injury whether or not the patient has been concussed. Some workers restrict chemoprophylaxis to those cases with rhinorrhoea, otorrhoea and open depressed fractures (Gurdjian and Webster 1958) but this is insufficient because most patients who develop meningitis never had a discharge from the nose or ear

A sulphonamide should always be given because it readily passes the blood-brain barrier and enters the cerebrospinal fluid Sulphadiazine is probably the drug of choice the dosage in adults is 4-6 g per day for 7-10 days or longer if rhinorrhoea or otorrhoea persist Penicillin intramuscularly or chloromycetin orally (not both, because of possible mutual antagonism) are often advised but they should be given in addition to and not as a substitute for sulphadiazine. Penicillin and the tetracycline drugs also should not be administered concurrently

The results of prophylaxis are difficult to evaluate because of the relative infrequency of meningitis after head injury but our impression, like that of Gurdjian and Webster (1958) is that the incidence has been reduced. Most cases of meningitis after head injury now seen by us are in patients transferred from peripheral hospitals whilst a few years ago before routine chemoprophylaxis was instituted, most cases occurred in patients brought directly to our hospital with a head injury

Chemoprophylaxis has its limitations and failures Meningitis occasionally develops after therapy has ceased or during its administration One of our cases of pneumococcal meningitis was diagnosed the day after a 5-day course of aureomycin had been completed and two others after a course of intramuscular penicillin. Meningitis developing during chemoprophylaxis is usually associated with penicillin therapy without a sulphonamide For example, a patient with a fracture of the frontal region extending into frontal and ethmoid sinuses was given 100 000 units of penicillin 6-hourly after admission but within 2 days he developed meningitis due to penicillin sensitive pneumococci Pneumococcal meningitis might even occur during combined penicillin-sulphonamide therapy or chloromycetin therapy one of our patients with transient cerebrospinal rhinorrhoea who was given 3 g. sulphamezathine and 0.5-2.0 million units of penicillin daily developed meningitis and two cases of pneumococcal meningitis during chloromycetin prophylaxis were reported by Collins (1956) In all these cases the pneumococci isolated were sensitive to the drugs administered prophylactically meningitis developed presumably because the antibiotic concentration in the cerebrospinal fluid was insufficient to inhibit the growth of invading pneumococci In other patients given chloromycetin or a sulphonamide the drug may modify but not prevent the disease

Symptoms may be masked and diagnosis prove difficult The spinal fluid contains many polymorphs but microscopy and bacteriological examination may fail to demonstrate any organisms This is one of the possible causes of abacterial pyogenic meningitis (see below)

SYMPTOMS AND DIAGNOSIS

In most cases meningitis develops within 7 days of injury but sometimes it may not begin until weeks months or even years later when the relationship to the head injury may easily be overlooked one of our cases occurred 5 years after injury and about 4 years after repair of the tear in the dura mater A common story is that the patient is admitted to hospital with concussion recovers completely within a few hours and is discharged the next day 1-5 days later he is readmitted with meningitis

Clinically the onset may be acute or insidious examples of the former mode of onset run a fulminating course with death within 24 hours either before therapy has begun or before it can reasonably be expected to have controlled the infection

About two-thirds of the patients have an acute onset with rapid development of classical symptoms and signs In them clinical diagnosis is usually easy particularly if the possibility of meningitis is considered The remainder have an insidious onset and course and clinical diagnosis may be difficult In these cases the classical signs of headache vomiting drowsiness neck rigidity and pyrexia come on at different times so that a few days may elapse before the picture is fully developed Indeed some patients have died with little more than pyrexia and headache as clinical evidence of meningitis Presenting signs such as convulsions acute hemiplegia or symptoms suggestive of encephalitis sometimes overshadow the early picture and make diagnosis difficult

There is a considerable polymorphonuclear leucocytosis in the blood and in some cases blood culture is positive for pneumococci This indicates that septicaemic invasion is always a danger

Differential diagnosis

The condition has to be distinguished from (1) meningism from (2) traumatic subarachnoid haemorrhage and (3) extradural haemorrhage and (4) subdural haematoma.

Meningocerebral irritation from subarachnoid haemorrhage may produce neck rigidity and a positive Kernig's sign and lumbar puncture may be necessary to decide the correct diagnosis The lucid interval between recovery from concussion and onset of drowsiness in cases of meningitis is usually longer than 24 hours but occasionally it is only a matter of hours and not unlike that of extradural haemorrhage Moreover a few cases of extradural haemorrhage are relatively delayed and do not develop until 1-3 days after injury so that the reliability of the duration of the lucid interval is relative and not absolute The combination of extradural haemorrhage and pneumococcal meningitis is rare but was found in one of our patients The possibility of subdural haematoma may give rise to difficulty in patients who become stuporose develop an unexplained pyrexia or of a head injury and who become stuporose develop an unexplained pyrexia or present unusual or unexpected symptoms particularly when concussion has been of short duration or absent and when the injury has involved the frontal or temporal area Lumbar puncture should then be performed

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Lumbar puncture

This must be carried out whenever meningitis is suspected. Diagnosis will be obvious if a milky or white turbid fluid is obtained but often the fluid is less characteristic. Blood in the cerebrospinal fluid may make a macroscopic diagnosis difficult or impossible. Laboratory examination is necessary and should include microscopic examination of a film of the fluid or its centrifuged deposit stained by Gram's method, culture for bacteria, a total white-cell count and protein concentration estimation. A tentative diagnosis of pneumococcal meningitis should be made when Gram-positive diplococci are seen and the patient must be put under treatment immediately. If numerous pus cells are present but no bacteria are seen it is wise to treat the case as one of pneumococcal meningitis at least until the results of bacterial culture (and if necessary mouse inoculation test) are available. When laboratory facilities are not immediately available the case should also be treated as pneumococcal meningitis without delay.

TREATMENT

The mortality of pneumococcal meningitis was little short of 100 per cent before chemotherapy was introduced and although the prognosis was improved by the use of sulphonamides it was not until penicillin was used that a considerable change took place. Now the survival rate of treated patients is generally about 70-80 per cent or higher if fulminating cases dying within 24 hours of instituting therapy are excluded. Early institution of treatment is of great importance and this depends on early diagnosis. Three lines of therapy have been used giving apparently equally favourable results and although most series reported are mainly of patients with pneumococcal meningitis unrelated to head injury the basis of treatment in cases following trauma should be the same. The alternative methods are as follows:

(1) Intrathecal injections of penicillin daily for 7-10 days supplemented by oral or intramuscular administration of sulphadiazine and perhaps by parenteral penicillin (Smith, Duthie and Cairns, 1946; Jepson and Whitty, 1946; Appelbaum, Nelson and Albin, 1949; Jackson, 1950; Gibson and James, 1952; Weinstein, Goldfield and Adams, 1953; McKendrick, 1954; Pan, Ting and Chang, 1959).

(2) Massive doses of penicillin given intramuscularly at 2-hourly intervals or by a continuous intravenous drip, preferably with sulphadiazine as a supplement (Lowry and Quilligan, 1948; Dowling and his colleagues, 1949; Lepper and Dowling, 1951; Burn and Peabody, 1952; Alexander, Flippin and Eisenberg, 1953; Gurdjian and Webster, 1958; Pan, Ting and Chang, 1959).

(3) Chloromycetin therapy (Parker and his colleagues, 1955).

Meningitis from streptococci sensitive to penicillin and chloromycetin should respond as well as the pneumococcal form. If the organism is insensitive or relatively insensitive to penicillin and sensitive to chloromycetin (as many nonhaemolytic and viridans streptococci are) therapy with chloromycetin will be necessary. It is therefore essential to culture the offending bacterium from the spinal fluid and to test its sensitivity to penicillin, chloromycetin and other antibiotics. This should not delay early commencement of therapy.

TREATMENT

Intrathecal penicillin supplemented with sulphadiazine

Therapy should follow the four principles advocated by Smith Duthie and Cairns (1946) namely (1) early institution of treatment (2) free access of adequate amounts of penicillin to all parts of the cerebrospinal pathway (3) maintenance of an inhibitory concentration of penicillin in the cerebrospinal fluid for a sufficient time and (4) the use of supplementary sulphadiazine. Intrathecal dosage varies in different series but is generally 15 000–50 000 units. Our experience has been mainly with this method of treatment and our practice has been to inject 20 000 units (10 000 units in children) freshly prepared in 2–5 ml of sterile saline solution every 12 hours during the first 1–2 days and then daily for at least another 7 days or until 2–3 days after the patient is clinically well and the cerebrospinal fluid has become relatively normal. Large doses of penicillin are toxic to the nervous system and should not be given. Convulsions collapse and death have been known to occur shortly after intrathecal doses of more than 100 000 units. Penicillin in concentrations of thousands of units per ml is toxic to skin in culture (Cruickshank and Lowbury 1952) so that its toxicity to brain is not surprising. Repeated intrathecal injections are necessary because penicillin diffuses out of the subarachnoid space although the rate varies from patient to patient. Diffusion is said to be delayed by systemic therapy. The penicillin level should be assayed on each specimen of fluid which is removed before penicillin is injected. The concentrations achieved must be effective and levels above 0.2 unit per ml. are satisfactory (this is 8–10 times the minimum inhibitory concentration against pneumococci) up to 2–5 units per ml may be found during the early period. Intrathecal injections are given twice daily for the first day or two because assay takes 24 hours or longer. Once satisfactory concentrations and response

Failure to continue daily therapy for 7–10 days is the cause of most relapses. This is a real danger if intrathecal penicillin is stopped after 2–4 days or if days are missed between injections.

Sulphadiazine plays an important role in therapy although it cannot by itself cure most infections. Dosage of 6–8 g. in 4–6 divided doses should be given for 14 days. Intramuscular administration may be necessary at first but later it is given orally. Concentrations between 8 and 14 mg. per 100 ml. of cerebrospinal fluid are usually obtained. Intramuscular penicillin is essential when pneumococcal septicaemia is present or when other pneumococcal lesions are present or suspected.

Changes in the cerebrospinal fluid

Within 24 hours (rarely 48 hours) of starting treatment the cerebrospinal fluid becomes sterile although pneumococci mostly degenerate forms are often seen in film preparations for 2–3 days. The initial cell count of hundreds or thousands per c.mm. often rises for 1–2 days and then falls. The early rise may be considerable. This is not the result of intrathecal injections as it may also occur during massive parenteral therapy with penicillin. As the total count falls the ratio of polymorphs to lymphocytes and macrophages decreases until no polymorphs are found. Therapy should be continued until this stage is reached although a residual count of 20–100 lymphocytes per c.mm. may persist for weeks. The protein concentration

MENINGITIS AFTER HEAD INJURY

may also show a temporary rise with treatment before it falls. Glucose is generally reduced or virtually absent at first but not in all cases.

Disadvantages

The major drawbacks of multiple spinal punctures are the discomfort to the patient, the possible danger of secondary infection, and the amount of medical and nursing attention required. Cases of arachnoiditis, spinal block and persistence of aseptic meningitis in earlier series have become rare now that pure preparations of penicillin are used.

Massive penicillin therapy

Massive penicillin therapy is preferably combined with sulphadiazine in full dosage because of the synergistic effect. The basis of treatment is that very large doses of penicillin promote some diffusion across the blood brain barrier when the meninges are inflamed so that inhibitory levels in the cerebrospinal fluid against pneumococci can be produced; this is not so with ordinary dosage. One million units intramuscularly every 2 hours has produced excellent results (Dowling and his colleagues 1949; Lepper and Dowling, 1951; Burn and Peabody 1952).

Concentrations of penicillin between 0.05 and 1.25 units per ml. in the cerebrospinal fluid were achieved whilst the blood levels were 20–100 times higher. Wide variation in dosage by different authors and differences in supplementary therapy were generally followed by similarly effective results, thus making recommendation of minimal or optimum dosage difficult but less important. The minimal amount of penicillin necessary for clinical cure is probably lower when sulphadiazine is also given. Thus daily dosages ranging between 400 000 and 2.4 million units combined with sulphadiazine or other sulphonamide were highly effective (Lowry and Quilligan 1948; Alexander, Flippin and Eisenberg, 1953). Pan Ting and Chang (1959) used 4 or 5 million units intravenously for the first 3 days followed by 2.5 million units daily of intramuscular procaine penicillin until the cerebrospinal fluid returned to normal, which was usually about 10 days. 8 of their 9 patients survived.

Continuous intravenous therapy using 25–34 million units every day in 3 l. of 5 per cent glucose solution as an alternative to 10–15 million units intramuscularly was advocated by Gurdjian and Webster (1958) for meningitis following head injury and they also used sulphadiazine. The survival rates in different series range between 70 and 80 per cent and the results are at least as good as those found with intrathecal penicillin.

It seems wise to give at least 10 million units of penicillin and 6–10 g. of sulphadiazine daily. Treatment should be continued for 10–14 days, or until 4–5 days after the infection has clinically abated. Some workers advocate a single intrathecal injection of 20 000 units of penicillin at the beginning to hasten an effective antibacterial level in the cerebrospinal fluid. Occasionally a course of daily intrathecal therapy may be necessary to cure relapses. Lumbar puncture should be carried out 24 and 48 hours after beginning therapy to ensure that the cerebrospinal fluid is sterile and, if all is well, it need not be repeated until 7 and 14 days later. An early rise in the white-cell count of the spinal fluid is frequent and not necessarily a cause for anxiety.

COMPLICATIONS AND SEQUELAE

One point of warning needs emphasis. Neither tetracycline antibiotics (aureomycin, terramycin, achromycin) nor chloromycetin should be given when patients are receiving penicillin because they have an antagonistic effect. Lepper and Dowling (1951) found that the mortality rate in pneumococcal meningitis was 30 per cent with parenteral penicillin alone but was 79 per cent when similar therapy was combined with aureomycin. Chloromycetin also antagonizes penicillin action and deaths following the combination have been reported (see below). Chloromycetin should not supplement penicillin therapy nor *vice versa*. This advice was also given by Hoyne (1953) who favoured massive parenteral penicillin therapy.

Chloromycetin therapy

Chloromycetin also diffuses into the cerebrospinal fluid from the blood when meningitis is present. It was used usually without other drugs by Parker and his colleagues (1955) they gave it in maximum dosage intravenously at first to achieve a prompt high concentration and then by a stomach tube or orally when it could be taken. Daily dosage to adults was 50-75 mg. per kg. body weight (3.5-5 g. for the average adult) and to children 100-125 mg. per kg. in divided doses. Seventeen cases were treated and there were 3 deaths (17.6 per cent). It is noteworthy that 5 patients were also given penicillin and 2 of them died. Parker and his colleagues pointed out that the optimum treatment of pneumococcal meningitis is not yet established and that treatment with chloromycetin should not necessarily replace massive penicillin therapy. Chloromycetin may of course be essential if the infecting organism is resistant to penicillin.

COMPLICATIONS AND SEQUELAE

Complications are now relatively infrequent and the majority of survivors recover completely. Cerebrospinal complications are relapse, spinal block, block within the cranium either without hydrocephalus (Fig. 68) or producing communicating or non-communicating hydrocephalus and rarely subdural abscess. Neurological sequelae are usually mental retardation or various cranial nerve palsies particularly deafness, optic atrophy and third nerve paralysis. Sometimes these may have resulted from the cerebral trauma rather than from meningitis. Repeated attacks and persistent rhinorrhoea are the major indications for dural repair. Repeated attacks are usually readily distinguished from relapses by the period of recovery between the attacks, otherwise a different serological type of pneumococcus indicates a new attack rather than a relapse.

A relapse must be treated with a full course of therapy as if it were a new attack. If spinal block develops during intrathecal therapy treatment with massive doses of penicillin or with chloromycetin should be immediately substituted. This may avoid the necessity of administering penicillin by ventricular puncture as was advocated by Smith, Duthie and Cairns (1946).

Causes of death

The major cause of death has been lack of treatment through failure to make a diagnosis or delayed institution because of delay in diagnosis. In treated patients

MENINGITIS AFTER HEAD INJURY

about half the reported deaths occurred within 24 hours of beginning therapy and most of them were fulminating cases. It is probably impossible to save the majority of these by chemotherapy and thus the survival rate is unlikely to exceed 85 or 90 per cent even when vigorous therapy is instituted at an early stage. Others die from a variety of causes some related to the meningitis, such as cerebral subarachnoid blockage with or without hydrocephalus (Fig. 68) and others from complications of other injuries like fat embolism or particularly in elderly patients from bronchopneumonia, cerebral softening and pulmonary thrombo-embolism. In these patients autopsy shows no evidence of active meningeal infection.

ABACTERIAL PYOGENIC MENINGITIS

Occasionally no bacteria can be demonstrated in the cerebrospinal fluid of patients with acute clinical meningitis after a head injury and fractured skull although the cerebrospinal fluid is turbid and contains hundreds or thousands of polymorphs per c.mm. Some cases can be explained by partial suppression of meningitis through prophylactic chemotherapy with sulphonamides or other drugs. In others this explanation does not suffice and 4 of our cases were of this kind. The cerebrospinal fluid leucocyte count was initially between 2 and 10 thousand per c.mm. (90 per cent polymorphs) organisms were not seen microscopically culture was sterile and negative results were obtained when mice were inoculated intraperitoneally in an attempt to isolate pneumococci. Three patients had cerebrospinal rhinorrhoea one of them aspirated air into the cranial cavity and the air encephalogram revealed evidence of considerable damage to the frontal lobes. The patients were treated with penicillin intrathecally and with sulphadiazine they recovered fairly quickly but this may have had no relationship to therapy.

The aetiology is obscure. In one case the cerebrospinal fluid glucose level was reduced but in the others it was relatively normal suggesting absence of bacterial metabolism. In 3 cases the possibility of certain viral infections was investigated serologically but complement fixation and other tests were negative for mumps lymphocytic choriomeningitis, influenza A and B, Q fever, psittacosis-lymphogranulomatosis group and for leptospiral infections. The dominance of polymorphs in the cerebrospinal fluid also made viral infection unlikely. The aseptic but pyogenic nature of the condition is consistent with a meningeal reaction from sterile necrotic brain substance or breakdown products of brain liberated after traumatic pulping. The evidence of considerable frontal-lobe damage in one patient is in accord with this. The meningitis may thus have been chemical and autogenous. A similar view was expressed by Cairns and his colleagues (1947) to explain the occasional cases of aseptic meningitis after open war wounds to the head.

REFERENCES

- Alexander, J. D., Filppin, H. F. and Eisenberg, G. M. (1953) *Arch. Intern. Med.* 91 440.
Appelbaum, E., Nelson, J., and Albin, M. B. (1949) *Amer. J. med. Sci.* 218 260.
Burn, P. A., and Peabody, G. (1952) *Arch. Intern. Med.*, 89 736.
Cairns, H., Calvert, C. A., Daniel, P. and Northcroft, G. B. (1947) *Brit. J. Surg. War Surg. Suppl.* 1 198.
Collins, D. L. (1956) *Canad. med. Ass. J.* 75 660.

REFERENCES

- Crickshank C N D and Lowbury E. J L. (1952). *Brit med J* 2, 1070
- Dowling, H F Sweet L. K. Hirsh H L. and Lepper M H (1949) *J Amer med Ass* 139 755
- Galloway W H and Chambers, W (1953) *Lancet* 2 68.
- Gibson, C D and James, D G (1952). *Lancet* 2 1203
- Gurdjan, E. S and Webster J E. (1958). *Head Injuries* London Churchill
- Hoyme, A L. (1953) *Med Clin N Amer* 37 329
- Jackson W P U (1950) *Arch Dis Childh* 25 22.
- Jepson R P., and Whitty C W M (1946) *Lancet* 1 228
- Lepper M H and Dowling H F (1951) *Arch Intern Med.*, 88 489
- Lowry G H., and Quilligan J J (1948) *J Pediatr.*, 33, 336
- McKendrick, G D W (1954). *Lancet* 2 512.
- P'an Po-Min, Ting Tsai-Tao and Chang Hsiao-Chih (1959) *Chin J Int Med* 7 55 (abstr in *Chin med J.*, 78 284)
- Parker R T., Snyder M J Ray S J L. Looper J W and Woodward T E. (1955) *Anti-
Bot Med.*, 1 192.
- Smith, H V Dunthie E. S. and Cairns, H (1946) *Lancet* 1 185
- Weinstein L. Goldfield, M and Adams, D (1953). *Med Clin N Amer.*, 37 1363

CHAPTER 18

HAND INJURIES

P S LONDON

INTRODUCTION

THE TWO main trends in the treatment of injured hands are first, the development of a new specialty concerned with the materials instruments and techniques of repair and reconstruction (Mason 1955) and secondly an increasing realization that it is more important to restore function than to conserve structure. Though injured structures can be saved by elaborate specialized measures these will have been misapplied if the result, however elegant proves to be of little or no use to the patient.

Hand, brain and mind

It is a truism that the efficiency of the hand depends not only on its structure but also on the brain and the mind that control it consequently the outcome of an injury depends upon the extent to which the hand brain mind unit is disorganized. For many purposes return of function to a level of feeling and movement below normal is compatible with full efficiency provided there is determination to succeed. The surgeon, therefore, must foster this determination as well as try to meet the individual patient's functional requirements. Sound judgment and simple measures may be better than technical virtuosity.

Elaborate programmes of reconstruction are necessary in some cases but carry the dangers that arise from prolonged incapacity and disablement. Morale and enthusiasm are liable to wane and a mental image of a more or less defective member may become established. Trick movements and new habits may be developed in place of the missing functions and the patient may find them easier than learning to use the reconstructed extremity. These influences rarely exist alone and are often abetted by anxiety and discomfort, sometimes by resentment and desire for gain. It is depressing to observe how a patient's initial confidence and resolution fade once he has left hospital and becomes exposed to the advice and opinions of people who may regard an early return to work as weakening a claim for compensation. The relationship and confidence between the patient and the surgeon should be developed as a shield against such influences.

Quite apart from their tenderness or awkward situation some patients are very anxious about scars especially skin grafts, to which may be attributed astonishing degrees of permeability to heat, cold dust, dirt, grease moisture and other supposedly noxious agents. Unnecessary bandages or finger stalls combine with timorous disuse to allow horny skin to accumulate and restrict movement by its rigidity and alter sensation by its thickness. In the face of such acquired disability small wonder that many patients persist in the belief that such fingers are not

right and must be further safeguarded Anticipatory explanation and reassurance is always indicated but does not always convince
This is the background against which treatment must be planned

PLANNING TREATMENT

The objective is to meet the patient's needs as far as the injuries will permit. It is necessary to decide in advance what is the most useful result that can reasonably be expected and then to decide how it can most effectually be achieved (Fig. 69). When more than one operation will be necessary each should be planned to facilitate its successor and if possible to allow the hand to be used in the intervals. The importance of prompt healing cannot be over-emphasized. Treatment cannot be planned until the full extent and severity of the injury has been determined.

Examination

As a general rule the value of clinical and radiological examination is inversely proportional to the severity and complexity of the injury. With closed injuries careful clinical and radiological examination commonly gives all the information required for diagnosis and treatment. With open injuries paradoxically diagnosis often cannot be completed until anaesthesia has been induced. The recently injured patient may have difficulty in co-operating reliably in the examination of motor and sensory function but even with trivial looking wounds damage to tendons and nerves may be indicated by simple observations of loss of normal tone and posture or absence of sweating. With children the response of the hand or fingers to the gentle contact of a hidden pin can be helpful. Even so exploration is often the only reliable way of being sure a partly divided tendon may function normally at first but give way a few weeks later. When therefore, a tendon is found exposed in a wound it should be made to traverse its full range in each direction in case movement since injury has carried a partial division under cover. Exploration and the factors governing the details of treatment are dealt with more fully below (see Open Injuries, page 283).

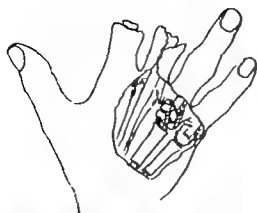
Anaesthesia and preparation

If only the hand is to be operated on a brachial plexus block is satisfactory but especially with long operations the patient should be rendered drowsy. When anaesthesia has been induced the surgeon himself should prepare the limb and at the same time make a more detailed examination. He can then decide what he is going to do if the need for skin grafting is foreseen. He can arrange the position of the patient and towels appropriately before settling down to an operation in which one or more donor areas may be required. There is much to be said for preparing the limb without the embarrassment of bleeding whether or not the operation itself is to be carried out with the circulation arrested.

Use of a tourniquet

Absence of bleeding greatly facilitates the operation and though speedy surgery is not justifiable as an end in itself the complexity of the injury the condition of

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(a)



(b)



(c)



(d)

FIG 69 —(a) Diagram illustrating an injury inflicted by machinery. *Assessment* — Index finger . trimming of existing stump. Middle finger . skin and bone too badly damaged for any to be worth saving. Disarticulated at metacarpophalangeal joint. Ring finger . finger as such undamaged but fracture of metacarpal bone and loss of skin and tendon will make it functionless even after elaborate repair. Skin better employed closing large gap on dorsum of hand. Little finger . main need is for restoration of tendon, reduction of fracture and closure of gap in skin. Skin supplied by the ring finger . tendon by the middle finger and fixation of fracture by wire suture. All this was carried out at the first operation and the patient began to use the hand after 2 weeks. (b) (c) and (d) Result 3 months later. Movement at metacarpophalangeal joint of little finger increased after extensor tenolysis.

CLOSED INJURIES

the patient or the presence of other injuries that need operation may make it desirable to avoid delay caused by bleeding, not to mention the loss of blood which may be considerable. Though the circulation has been arrested for several hours without evident damage it is a wise intention not to leave a tourniquet on for more than an hour to an hour and a half which is long enough for many operations to be completed and a haemostatic dressing applied. With longer operations bleeding at some stage is inevitable and if a tourniquet is to be used it is perhaps most helpful at the beginning of the operation when it facilitates detailed exploration of the wound and preparation for subsequent repair. Twenty or thirty minutes often suffice for this and such a period of anoxia is not followed by much reactive bleeding. To plod on with an operation and remove a tourniquet only when the voice of prudence can no longer be ignored results in bleeding that varies from at least a persistent widespread ooze to an alarming torrent.

A rubber bandage can safely be wound round the limb to exsanguinate it but an air-cuff is preferable for maintaining circulatory arrest. It should be firmly secured and the pressure raised to at least 300 mm of mercury. Someone else should be made responsible for ensuring that this pressure is maintained but the surgeon must be responsible for seeing that it is released.

Perhaps the strongest argument against using a tourniquet is the fact that it does not allow one to distinguish between tissues that do and do not retain a circulation. This objection is not always relevant.

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Crushing

The effects of crushing, which range from mild bruising to severe and lasting stiffness can be due to disruption and subsequent scarring of gliding tissues and occur apart from fracture. Radiography should not be omitted lest a trivial but unsuspected fracture come later to light and provide grounds for a charge of negligence.

Painful haematomas require drainage and if large as may occur in the palm call for evacuation through an incision. Aspiration is not feasible until 10-14 days have passed. A more severe type of tension injury though not strictly a closed one is the rare grease-gun injection. The tissues of the hand are more or less widely infiltrated by grease, which is difficult or impossible to remove completely and even in small quantities may give rise to a foreign body reaction with persistent induration and disability. Repeated operation may then be required. Massive injection can cause ischaemic necrosis by its bulk alone.

Sprains

Genuine sprains that is mild capsular injuries are not uncommon and usually do well if protected. An injured finger is hitched to its neighbour by strapping or a garter the thumb is strapped. Not all digits that are swollen, stiff and painful after injury have merely been sprained, however. In some there has been subluxation with complete rupture of more or less of the capsule and perhaps avulsion of a chip of bone. Lateral instability of the affected joint should always be sought and may require operative repair. This is particularly important in the thumb in which usually the postaxial side of the capsule of the metacarpophalangeal joint

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is torn (Frykman and Johansson 1957). Laxity here cannot be overcome by muscular control and it causes persistent weakness and discomfort in the gripping between finger and thumb. The key lesion is a tear in the sheet of capsule between the collateral ligament and the extensor hood. Chronic laxity of the joint can also occur after repeated lateral stresses and has been described as gamekeeper's thumb (Campbell 1955).

Another diagnostic trap is the dislocation or fracture-dislocation that has been reduced, perhaps spontaneously, before the patient reaches hospital (see below). It is advisable to diagnose a sprained digit only after clinical and radiographic examination has ruled out more serious damage.

Dislocations

Many dislocations can be reduced easily without anaesthesia and require more support than sprains, but two groups require operation. In one group a part of the capsule prevents closed reduction. Either the fibrocartilaginous palmar ligament is displaced behind the head of the proximal bone or the head of the proximal bone erupts through its surrounding structures, which then grip the neck of the bone ("buttonholing"). This usually occurs at the first metacarpophalangeal joint where the buttonhole is bounded by the interosseous tendon on one side and the tendons of the long flexor and the thenar muscles on the other.

The other group is less well known and includes some dislocations with associated chip fractures. The dislocation is reducible by manipulation but should always be radiographed again as the fracture may be recognizable only after this. In fingers the proximal interphalangeal joint is most often affected and the piece of bone is torn from the flexor lip of the middle phalanx (Fig. 70). Provided this returns to its rightful position the joint requires protection only, but should remain well away from its source, the chip, which has the palmar ligament attached to it, needs open reduction and fixation by fine stitches that may be tied locally. It may be of the pull-out variety (Moberg and Stener 1954). Otherwise the joint tendons become matted by scar that resists physiotherapy and remains undisturbed by capsulectomy.

In other cases the chip comes from one side of the base of the phalanx and should be replaced if it remains much displaced or rotated.

Fractures

Fractures may be divided into three groups: those that require neither manipulation nor fixation, those that can be treated by closed reduction, and those that require operation.

Manipulation not required

The fractures themselves may need no treatment but this does not apply to all patients who often require explanation, reassurance, demonstration and persuasion before they leave the hospital, sometimes without even a bandage, or a plaster-of-Paris. Spiral fractures of metacarpal bones, most fractures of the neck of the fifth and shaft of the first metacarpal bones and simple fissures in the phalanges, are in no danger of being displaced by use that the patient will tolerate. The initial discomfort caused by movement may make rest seem advisable but often swelling and painful stiffness are reduced by gentle activity.

CLOSED INJURIES

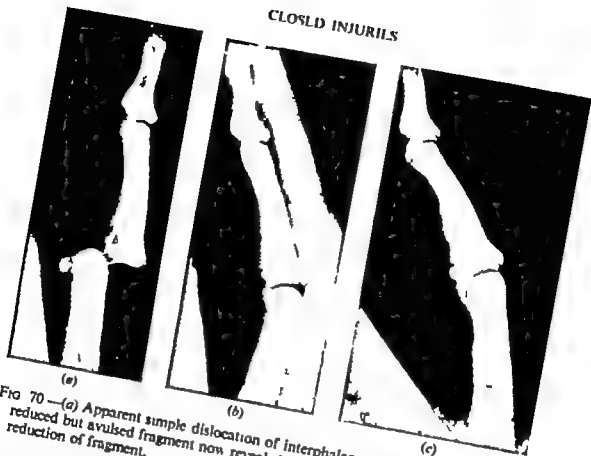


FIG 70—(a) Apparent simple dislocation of interphalangeal joint. (b) Dislocation reduced but avulsed fragment now revealed and widely displaced. (c) After open reduction of fragment.

The various types of plaster recommended may rest the hand by encumbering it, but do not add usefully to the security provided by muscles or impaction. Furthermore attempts to correct deformity by plaster often fail and may cause pressure sores.

Closed reduction

Closed reduction is effective for many mobile fractures with displacement. Deformity can often be corrected by pulling on the digit and fixing it in flexion on a palmar splint of plaster or Kramer wire or by strapping it over a roll of bandage in the palm. Whatever method is used the line of the finger must extend towards the base of the thenar eminence. Occasionally a fracture with extension angulation is irreducible except by operation because a spike of bone has penetrated the sheath of the flexor tendons. Lateral angulation such as commonly occurs with injuries of the proximal phalangeal epiphysis can be treated least cumbrously by manipulation and hitching the finger to one or both neighbours. Enclosing more or less of a finger particularly a marginal one in plaster may cause angulation at the fracture (Fig. 71).

Open reduction

Open reduction is needed for fractures that are unstable or irreducible by manipulation. Its best justification is not that it shall restore anatomical relations but that it shall allow early use. It should be regarded as part of the treatment of the soft tissues and moving parts.

HAND INJURIES

Displaced transverse fractures of phalanges or metacarpal bones, especially when multiple are perhaps most effectively dealt with by intramedullary pegs of bone though these are less easily inserted than wire sutures or Kirschner's wire. It is not always necessary to fix each of several fractures. Transfixion is convenient and effective with unstable oblique fractures. The wire must fit tightly and if cut part way through at the right place before insertion it need not project beyond the bone.

Bennett's fracture-dislocation gives very good results when the small fragment is large enough to take a fine ($\frac{1}{16}$ inch) screw. In other cases active supervised exercise from the beginning restores a good range of movement but the patient must be prepared to accept some weakness. Various methods of transfixion and manipulation followed by a carefully moulded and padded plaster case have their advocates (see Chapter 10).

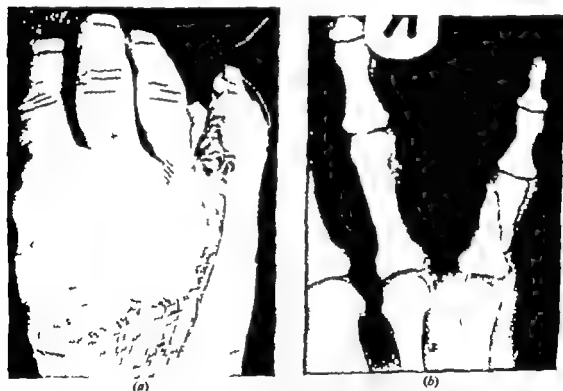


FIG 71—(a) Basis of type of plaster commonly used for phalangeal fractures. (b) Angulation caused by plaster of this kind.

Injuries of tendons

The commonest closed injuries affect the extensor tendons at one or other interphalangeal joint and there may be an avulsion fracture as well indeed without this the torn middle slip often passes unrecognized until the lateral slips have separated enough to produce the characteristic deformity. The torn middle slip needs surgical repair. Mallet finger is usually better without it. The traditional method of immobilizing a mallet finger in the corrective position may fail because the plaster breaks, softens or is applied too loosely or because it is too tight and

OPEN INJURIES

has to be removed. In any case the corrective position cannot be relied upon to appose the edges of the tear. Few people are handicapped for long by a mallet finger but all are grateful for support during the early painful stage. A light removable metal or plastic splint keeps the injured joint at rest. The original deformity commonly lessens a little with time but operation may be called for in severe cases. The presence of a fracture usually gives direct suture a good prospect of success, otherwise a stiff but not always rigid joint is likely. Closed rupture or detachment of flexor tendons is rare in the absence of degenerative conditions such as rheumatoid arthritis and is treated along the usual lines.

Injuries of nerves

The digital nerves occasionally suffer partial division by crushing. The patient is more likely to complain of a painful neuroma than of impaired sensation and if the neuroma cannot be relieved of pressure by mobilization of the nerve simple excision should be considered in preference to secondary suture or grafting.

The deep branch of the ulnar nerve crosses the hook of the hamate bone and is here subject to pressure by for example the end of the handle of a lawn mower or of the handle bar of a motorcycle (Russell and Whitty 1947). This nerve can also be compressed by ganglia and other swellings in the hypothenar muscles (Seddon 1952). The condition is treated by removing the cause and is easily diagnosed if borne in mind. The patient complains of weakness with wasting restricted to the muscles supplied in the hand by the ulnar nerve.

OPEN INJURIES

Rank and Wakefield's (1953) separation into tidy and untidy wounds is apt and practical. Tidy wounds are inflicted by sharp objects that divide tissues and indeed, may sever them completely but otherwise usually do them little harm. Unless severe bacterial contamination has been likely these injuries can be expected to heal without infection and often there is a chance of completing the necessary repairs in one stage. Untidy wounds, on the other hand are inflicted by forces that crush, tear and disrupt the tissues adding destruction and devitalization to mere solution of continuity. Foremost among the unknowns that untidy injuries implanted in the wound. The patient complains of weakness with wasting restricted to the muscles supplied in the hand by the ulnar nerve.

Exploration

Unless the existing wound enables the surgeon's eye and not just an instrument to search its full extent it must be enlarged. Quite apart from facilitating inspection however it is often desirable to enlarge a wound so that structures in its depths can be dealt with comfortably without the need for strenuous and prolonged retraction that may jeopardize healing. Extension should err on the side of generosity but must always respect the blood supply of the skin nearby and if secondary operation in the area is foreseen the primary incision should be designed to embarrass it as little as possible.

HAND INJURIES

Though the rules governing surgical incisions may be violated by the wound the surgeon must respect them and his extensions of the wound must make for and continue along the accepted lines. When circumstances permit the scar of an awkwardly placed wound should be moved to a better position by means of a Z plasty or similar procedure again with due respect for the local blood supply.

Because tidy wounds can usually be relied on to heal well and thus to permit more or less complete repair it is reasonable to consider the management of the injured structures one by one.

MANAGEMENT OF INJURED STRUCTURES

Skin

No effort should be spared to secure reliable closure of healthy skin for this is the best barrier to bacterial invasion. Often no more than simple suture is needed and unless the edges of the wound are ragged or bruised they need not be trimmed. When damaged skin has to be cut away this should be done as sparingly as possible unless there is already a defect that can be closed only by grafting. It may then be advisable to sacrifice normal skin for the sake of moving awkwardly situated scars to places where they will not give trouble.

There is so little spare skin on the hands that even on the dorsum there is little scope for closure of gaps by simple advancement. Suture under tension is inadmissible and the position of rest should not be lightly abandoned for the sake of enabling a wound's edges to be brought together. There need be no hesitation over the use of free skin grafts to avoid these difficulties but flaps are more fickle and should not be used without good reason.

Severed finger-tips

Small pieces are often worth replacing and, particularly in children, much larger parts of the tip and pulp may be sewn back if they are still attached. Otherwise the choice lies between amputation with closure by free grafts, local flaps, and flaps from elsewhere.

Amputation might seem to offer a serviceable hand in the shortest time but this is by no means always so. Much of the trouble is due to tenderness of the stump and it must be admitted that the surgeon is often to blame. The long standing surgical tradition that every scrap of viable tissue in the hand should be retained has unfortunately not yet been entirely discarded. Many a stump that has been closed with the least possible sacrifice of tissue has thin shiny skin, bereft of fatty padding and sensation and stuck to awkwardly shaped projections of bone. The entire remnant is a burden. It is far wiser to shorten the digit enough to allow normal skin to close easily over the bone and provide a comfortable stump.

Free grafts of whole or thick split skin are suitable if bone is not exposed or if it can be nibbled away a little to allow the subcutaneous tissues to cover it, but if the terminal phalanx is shortened by more than a very few millimetres the nail curves downwards and its appearance may distress the sensitive. This can be avoided by the use of a thenar flap properly used in carefully selected cases this gives good results. Patients should be young enough to have supple joints and the

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stump must be long enough to reach the base of the thenar eminence without strain. The appearance is excellent but sensation remains defective. A troublesome scar on the thenar eminence is usually the fault of the surgeon. A graft from the inner surface of the lobule of the pinna is an ingenious source of padded skin but has no great advantage.

The tip of the thumb can be made good from the side of the fore or middle finger and a defect on the shaft of one finger can be filled by a flap when required from the back of a neighbour. The only other source of manual skin is a filleted digit on a short functionless amputation stump. When the demand is more than the hand can meet distant flaps have to be used. Except in the skinny flaps from the other arm or forearm are more suitable than those from the chest or belly. Valuable as flaps are the place for them is easily exaggerated by technical enthusiasm. Unless they offer clear advantages over amputation or free grafts they should be avoided.

Primary suture of tendons

Primary suture may be considered in three degrees of advisability

Almost always

Extensor tendons and flexor tendons cut in the palm should almost always be primarily sutured. Provided the flexor profundus muscle is intact there might seem to be no need to repair the flexor sublimis muscle. In fact, unless the distal segment of the superficial tendon is removed completely it may stick to its fellow and reduce movement. Competent suture is less disturbing. When both flexor tendons have been divided it is unwise to repair both unless the suture lines can be separated from each other without putting the finger in an awkward position.

Sometimes

The flexor profundus muscle may be cut distal to the flexor sublimis muscle. Few surgeons practise tendon grafting solely to restore movement at the tip of a finger. Primary suture may succeed but if it fails because of adhesions the tip is at least stable and this is certainly no worse than an uncontrollably movable joint. Repair however is inadvisable when contamination is likely to have occurred and if both palmar nerves have also been divided amputation is often the best treatment.

Tendons may at times be worth sewing up not to restore movement but to give added support while a badly damaged bone or joint is fusing.

Almost never

Primary suture should hardly ever be attempted when both tendons are cut in their sheath. The usual practice of closing the skin and inserting a graft some weeks later is safe. Primary suture and primary grafting have succeeded but are not recommended as a policy.

Treatment of nerves

Because they contain no motor fibres and run only a short course digital nerves are liable to be regarded as being singularly suitable for primary suture. In fact

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regeneration is often poor partly because of the technical difficulties of handling such small structures and partly because of uncritical selection of nerves for primary suture. Fortunately if regeneration does not occur after one nerve has been injured sensory overlap usually makes the eventual loss of sensation negligible, but when an otherwise valuable digit has been rendered useless by division of both palmar nerves suture or grafting offers the only chance of improvement. There may be a place for plasma clot union in the repair of these small nerves (see Chapter 20).

Treatment of bones and joints

An open joint with loss of skin may be one of the reasons for using a flap, but if the damage has been so severe that there is no chance of restoring movement, *excision and fusion of the joint is a useful procedure and by shortening the length of bone it may enable a gaping wound to be closed without resort to a flap.*

The case for internal fixation of fractures in reparable tidy wounds is based on the need to safeguard other structures, the desirability of early movement, and the advantage of facilitating necessary secondary repairs. The management of a wound that severs skin, tendon and bone, for example, is rendered much easier if the bone can be fixed: this eliminates the risk that it will become displaced and disrupt an adjacent suture line. It also safeguards contours over which a tendon will have to move. In other cases, amputation is the only practicable course.

Amputation

Much of the argument that has raged around this subject seems to have been due to a failure to appreciate clearly the purpose of amputation. It is to retain the most useful remnant in the face of irreparable damage. Usefulness of a stump depends first and foremost upon its being comfortable to use, but length, strength, mobility and sensibility, the presence and condition of other digits, and the particular patient's needs must all be taken into consideration.

It cannot be stressed too often that the closure of amputation wounds is no different from that of others: the tissues must be healthy and there must be no tension. These desiderata are set aside most often after traumatic amputation for the temptation to make do and conserve is then especially strong.

The following rules are widely applicable to single injured digits but have to be modified when several have been damaged.

Thumb—Preserve as much as will be useful.

Index and little fingers—Anything less than one and a quarter phalanges is usually a hindrance and should be discarded. For a strong hand, retain the head of the metacarpal bone; for elegance (according to taste) remove it.

Middle and ring fingers—Even short stumps are useful as stopgaps and the metacarpal bones should be retained when strength and span are particularly required.

In all fingers, amputation through the terminal phalanx is followed by more or less troublesome deformity of the nail that remains.

UNTIDY AND DESTRUCTIVE WOUNDS

The feature common to most untidy wounds is more or less widespread damage to the tissues: apart from this they vary so much that they do not lend themselves to

UNTIDY AND DESTRUCTIVE WOUNDS
orderly consideration nor can their management be reduced to a set of simple rules. One rule however may be laid down. Prompt healing outweighs in importance any other objective of initial treatment.

The first operation

The first operation is of fundamental importance and its prime purpose must be to lay sound foundations for further treatment. If it achieves nothing more than healing by first intention those foundations will have been well and truly laid for given prompt healing which also means absence of infection stiffness and deformity can be prevented or rapidly overcome and reconstructive operations commenced without delay and in the least unfavourable conditions. This is not to say that there is no place for definitive and even complete reconstruction at the first operation far from it. Indeed this is the ideal objective but not one to be pursued unless the tissues that are used can be relied upon to heal promptly. Herein is the crux of dealing with untidy injuries for in many cases the state of the tissues at the outset provides no trustworthy indication of their eventual fate and one that was originally adequate may be brought to a halt hours or even days later. Nevertheless a decision must be made and acted on without delay for there is no place for hopeful expectancy except upon a basis of well judged surgery. Broadly speaking, the first operation should be planned to conserve only those tissues that seem likely not merely to survive but also to be capable ultimately of serving a useful purpose. All else should be discarded. This may seem a ruthless policy but if properly applied it is not a reckless one. Its correct application is guided by principles rather than governed by rules and must be directed towards retaining as much of the basic functions of the hand as the circumstances permit.

Basic functions of the hand

The hand is a sensory organ. It can be used as a clamp to press upon and steady an object (Furlong (1957) has called it a paper weight). It can be used as a vice for powerful gripping, and as a forceps for precise grasp. The hand can be used as a clamp if there is no more than the base of the metacarpus. Anything more is advantageous. To act as a vice the hand needs at least one mobile digit and its metacarpal bone. Anything more is advantageous. To act as a forceps the hand needs a thumb and one other digit that can be approximated. Anything more is advantageous. Clearly then provided sensation is not seriously unpaired the hand can be effective for most common purposes if it possesses no more than a bone. Any other remnants are advantageous but only if they augment the basic functions. Now that so much can be done to restore injured hands it is important to recognize that the existence of a method of reconstruction does not of itself justify the application of that method. Elaborate surgical programmes are often necessary but for a working man they cannot be justified if simpler methods will restore adequate function more quickly.

Amputations and comparably destructive injuries

A single badly injured finger is unlikely to contribute usefully to the hand even after reconstruction and is usually better amputated at the outset. When several

or all the fingers have been badly damaged as much as possible of each should be saved at first two or three weeks later it will have become clear what is amenable to reconstruction and what must be discarded. Traumatic amputation of several fingers can be treated by multiple flaps but the patient's needs may be met equally well by say a thenar flap for one or at most, two fingers and free grafts or trimming for the other stumps. If more than two flaps are required they must be raised from the chest or other arm. Provided the thumb is essentially intact even complete loss of all the fingers gives a result upon which the surgeon is unlikely to improve but phalangeal remnants should be retained. The length of the metacarpus must be preserved and if there is not enough local skin for closure a flap will have to be brought in from elsewhere.

The practice of constructing digits from skin tubes and bone grafts has so many shortcomings as to be rarely worth while. The new digit is merely a rigid projection with no sensation and a poor circulation it is easily injured and ulcerates more readily than it heals. A missing thumb can be replaced in this way but provided there are at least three normal fingers a much better result is achieved by transplanting one of them (Littler 1953) or by rotation osteotomy to restore a pinching action. The hallux has been transplanted but not all patients have regarded the new and ungainly thumb with as much satisfaction as has the surgeon. Loss of the entire thumb is generally regarded as a crippling disaster but this is not always so and reconstruction is not obligatory.

More extensive injuries that affect the metacarpal bones as well as the phalanges are treated along lines similar to the above and there should be no hesitation in the use of free or flap grafts to ensure prompt healing and the preservation of important structures.

MANAGEMENT OF INJURED STRUCTURES

Badly Injured skin

The skin of a hand caught in a power press or between rollers suffers severe damage though the two types of machinery inflict fairly distinct types of injury. The power press crushes, splits and mangles to produce injuries that may seem at first to offer no alternative to further sacrifice of tissue for the sake of tidy amputation. It is usually necessary to remove some tissues but it is sometimes surprising how much can be saved by painstaking suture of tattered but viable remains (Fig. 72).

Rollers cause injuries which may look much less severe but the flaps of skin that they tear from the hand have been stretched and crushed in the process. They are usually based distally and their venous and lymphatic drainage is seriously embarrassed as a result. Such flaps are unlikely to survive to any useful extent and if sewn back may predispose to infection. In most cases, therefore flaps of this kind are better cut away at the outset. When there has been no loss of skin however there is something to be said for sewing flaps back if the survival of the underlying tissues is in doubt the flap may die but infection is by no means inevitable and dead skin is a useful dressing if it can be kept dry. If skin has been lost, grafting is inevitable and whatever type may be best in the long run, split-skin grafts are preferable to start with especially if the viability of the bed is suspect. In a number

UNTIDY AND DESTRUCTIVE WOUNDS

of cases split skin proves to be durable enough for permanent use and if care has been taken at the beginning to arrange scars to lie away from tension lines further operations can be avoided.

Patients can sometimes be spared the discomfort and inconvenience of having flaps raised from the chest or other arm if an injured finger is filleted and its skin used to cover a defect over the metacarpus or on another finger (Fig. 69). Further more an otherwise useless finger or amputation stump may be worth retaining at first so that its skin shall be available later to make good a defect or replace a troublesome scar foreseen at the outset. Skin transplanted in this way retains its nerve and blood supply.

Bones, joints and tendons

Primary repair is in most cases either impossible because of the extent or severity of the damage or inadvisable because of doubt about viability and healing but with care quite a lot can be done to preserve the injured structures in a useful state and even to eliminate the need for further operations. Whenever possible unstable fractures should be fixed so as to safeguard overlying suture lines and grafts and to allow early movements. There need be no hesitation about tidying a smashed joint and performing primary arthrodesis indeed the resulting loss of bone may enable gaps in overlying skin to be closed by simple suture instead of by means of a flap. Suture of tendons cannot often be expected to result in movement but it does add useful reinforcement and stability to the repair.

Pseudarthrosis is not recommended in the digits but the smashed head of the third or fourth metacarpal bone can be removed and leave a useful finger. The heads of the second and fifth bones however should not be discarded lest the finger subluxate away from its neighbour. The use of a graft or even a prosthesis should be considered if some of the bone has been lost. Gunshot and other penetrating injuries can wreak havoc among the metacarpal bones while the tendons, being tough but yielding, may escape serious damage and act as a shield for the neurovascular bundles. Once the wound has healed the missing bone can be made good by a single block or by individual grafts from the ilium and if as may happen the metacarpophalangeal joints are intact so much the better otherwise permanent stiffness must be accepted.

Muscles

Serious damage to muscles usually accompanies fractures but the thenar group can be damaged apart from bone by explosion or by crushing. In the first instance the muscles are destroyed often with some skin in the second some or all of the thenar muscles are extruded from a split in the first web. Though they retain one attachment to bone and bleed and twitch when pinched the extruded muscles should not be replaced for they become scarred and draw the first metacarpal bone down to the second. In each case the loss of opposition must be dealt with by transplanting a tendon or inserting a bone graft. It may also be necessary to re-form the first cleft by rearranging the available skin or augmenting it by free graft or flap.



FIG 72.—See legend on opposite page.

CARE AFTER OPERATION



(c)

FIG. 72.—The result of careful trimming and suture (a) At first sight there seemed to be no alternative to amputation at all four metacarpo-phalangeal joints. The skin edges were largely viable though ragged and careful trimming followed by painstaking suture with fine materials enabled useful stumps (b and c) to be retained.

CARE AFTER OPERATION

Dressings

The surgeon's attention should not be withdrawn when the last stitch has been inserted: the dressing can make or mar a successful operation and requires special care when applied before a tourniquet has been removed. Firm even pressure combined with a good functional position is effectually achieved by the use of a splint dressing of wet or greased wool. A large block placed in the palm is liable to keep the metacarpophalangeal joints extended. A determined effort should be made to fix them flexed to a right angle. Padding should be placed between digits. The hand should be elevated. In some cases of simple closure of skin and securely fixed fractures the dressing can be removed after a day or two and movements started. Grafts are better rested for a few weeks. It is often wise for the surgeon to carry out subsequent dressings himself for then he can see whether things are going according to plan and take prompt advantage of favourable developments or prompt steps to mitigate unfavourable ones.

Physiotherapy and reablement

The surgeon should be at least as much concerned to prevent swelling and stiffness as the physiotherapist to treat these conditions: but it takes almost super-human efforts to prevent them when a wound extends most or all of the way round

HAND INJURIES

a part. Whether the skin be sewn back or replaced by any sort of graft at any stage the disruption of lymphatic and venous pathways causes swelling beyond the breach.

Instruction and supervision of movement is the main need. Passive and assisted movements, massage, lively and other splintage, electrical treatment, and the various means of heating the tissues are also valuable but tend to confirm the patient's tacit belief that after his hand has been injured it must be treated (by other people) before it can be used (by him) (Ling and O'Malley 1958). The less time a man spends away from work the more likely he will be to regard the injured hand as something to be trained and used and not merely something to be treated. When treatment is necessarily prolonged every opportunity should be taken to keep the patient occupied between periods in hospital. His usual work or something like it may be within his powers but failing this a period at an Industrial Rehabilitation Unit should be considered. Light work is often a euphemism for idleness and should be regarded with suspicion. It may be difficult to persuade a person that work is treatment but the co-operation of employers can be of decisive importance. There should be no hesitancy about approaching them and the results are at times gratifying.

REFERENCES

- Campbell, C. S. (1955) *J Bone Jt Surg.*, 37B, 148.
Frykman, G. and Johansson, O. (1957). *Acta chir scand.* 112, 58.
Furlong, R. (1957). *Injuries of the Hand*. London: Churchill.
Ling, T. M., and O'Malley, C. J. S. (1958) *Rehabilitation after Illness and Accident*. London: Baillière, Tindall and Cox.
Little, J. W. (1953). *J plast reconstr Surg.*, 12, 303.
Mason, M. L. (1955). *Int Abstr Surg* 101, 541.
Moberg, E., and Stener, B. (1954) *Acta chir scand* 106, 166.
Rank, B. K., and Wakefield, A. R. (1953) *The Surgery of Repair as Applied to Hand Injuries*. Edinburgh: Livingstone.
Russell, W. R., and Whitty, C. W. M. (1947) *Lancet* 1, 828.
Seddon, H. J. (1952). *J Bone Jt Surg.*, 34B, 386.

CHAPTER 19

FACIOMAXILLARY INJURIES

R O WALKER

DURING World War II major advances in the management of faciomaxillary injuries took place and since then there have been only a few minor departures in methods of treatment.

A general review of the present position is presented here because the surgeon who is responsible for the general care and treatment of injured patients will only be concerned to a limited extent with the detailed and specialized nature of treatment which is usually deputed to the dental consultant and technicians trained in this work but others do not and in them the existing staff must treat both simple cases and those cases in which the pressure of other injuries precludes for a time at least transfer to a specialized unit.

Faciomaxillary surgery includes the surgery of the soft tissues of the face the facial skeleton and associated sinuses, the orbits and the anterior fossa of the skull but since most of the advances in treatment are related to the treatment of fractures of the jaws and their complications this chapter is directed primarily to a review of this aspect of the problem.

The mandible

A study of the architectural structure of the mandible reveals potential weaknesses in the condylar neck and in the tooth bearing segments particularly if the teeth are unerupted misplaced or the seat of pathological changes. The common sites for fractures in order of frequency are condylar neck angle of mandible (associated with erupting wisdom tooth) body of mandible (molar region) incisor region and canine area.

The displacement of the fractured bones is greatly influenced by the line of fracture and by muscle attachments as well as by the direction and force of the blow. A few fractures will be discussed in more detail to illustrate the problems of displacement which may arise.

Fractures of the condylar neck

Fractures of the condylar neck usually occur from indirect violence applied in the region of the symphysis menti.

The head of the condyle is usually displaced forwards and medially by the pull of the external pterygoid muscles (Fig. 73). Diagnosis is based on failure to palpate movements of the condylar head gagging of the occlusion on the molar teeth, and deviation of the mandible to the affected side. The displaced condylar head is inaccessible for reduction except by open operation but fortunately union in its misplaced position or even non union and a false joint impair function very

little. The displacement is therefore generally disregarded and treatment based on the maintenance of normal occlusion, voluntarily in simple cases or by inter maxillary fixation for 2-3 weeks in others. Thereafter active movements are encouraged provided that normal relationship of the teeth can be achieved and maintained.

Bilateral condylar fractures—Bilateral condylar fractures present special complications. The distracting effect of the condylar heads is lost as a result of their displacement and the elevator muscles acting on the ascending ramus tend to produce bilateral gagging of the occlusion on the molar teeth with an anterior open bite. This is aggravated by the action of the depressor muscles of the mandible attached to the symphysis menti.

This deformity can only be reduced with difficulty by the patient and if inter maxillary fixation is indicated it is usual to attempt to overcorrect the anterior open bite. A resultant small open bite between the molar teeth tends to be self correcting with use but to ignore the anterior open bite may lead to permanent disability.

Complications from derangements of the temporomandibular joint are rare and usually follow overimmobilization and restriction of movement.

Fractures in the region of the angle of the mandible

With fractures in the region of the angle of the mandible the main problem is the tendency for the posterior fragment to rise forwards and lingually so that if the fracture line is unfavourable, considerable displacement may occur (Fig. 73c and d). In the majority of cases immobilization of the main fragment and the splinting influence of the masseter and internal pterygoid muscles will provide control but on occasion direct control of the posterior fragment is necessary.

Fractures of the body of the mandible

When the fracture lies in the body of the mandible in advance of the attachments of the internal pterygoid and masseter muscles, immobilization can be difficult if no teeth remain on the short posterior fragment. The influence of the masseter and internal pterygoid muscles now aggravates any tendency to upward and forward displacement of the posterior fragment caused by the temporalis and external pterygoid muscles.

Bilateral fractures—When the fracture lines are unfavourable, bilateral fractures present special features and constitute one of the most difficult problems in treatment encountered in the mandible (Fig. 73e and f). The central fragment falls inwards, acute embarrassment to the airway may result from the loss of tongue control. Early treatment becomes a matter of urgency and any means of upward and forward reduction of the central fragment may be life-saving. Permanent fixation must be rigid and retained for longer than usual because of the unfavourable strains from muscles attached to the fractured components.

The middle third of the face

Fractures to the bones of the middle third of the face are less frequent than fractures of the mandible. They present a very different problem since they are associated, as a rule with the more severe forms of crushing injury. The complex

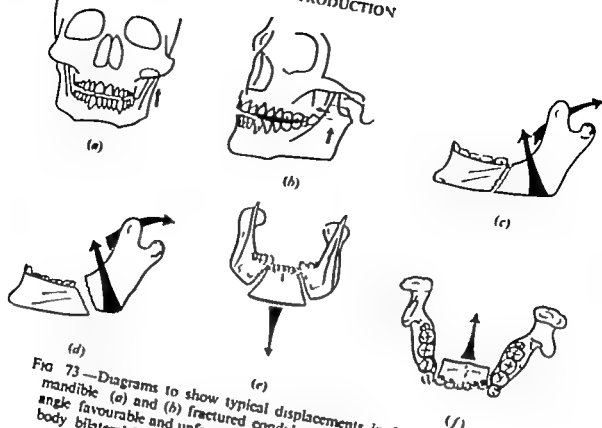


FIG 73—Diagrams to show typical displacements in fractures of the mandible (a) and (b) fractured condylar neck (c) and (d) fractured angle favourable and unfavourable fracture lines (e) and (f) fractured body bilateral fractures

structure and arrangement of the bones leads to possible involvement of the anterior fossa of the skull the orbit or paranasal sinuses (Fig 74a and b). Fractures of the nasal bones and zygoma are almost a commonplace their treatment is usually straightforward. Injuries to the central or dento-alveolar component require detailed discussion.

These fractures have been classified according to their site as follows: (a) alveolar fractures (b) subzygomatic fractures—(i) low level fractures (Le Fort I or Guerin's fracture) (ii) pyramidal fractures (Le Fort II fracture) and (c) suprazygomatic or high level fractures (Le Fort III fracture) (Fig 74a). These fractures occur along various suture lines so that while this classification is helpful variations and combinations may occur particularly if the injury is to one side. The levels of fractures may then differ on the two sides.

Le Fort I (Guerin's) fracture

The fracture line runs transversely above the apices of the teeth below the zygomatic processes and involves the antrum, floor of nose and the pterygoid processes. When complete it is characterized by mobility of the fractured fragment, which may come to lie on the dorsum of the tongue. This and the associated oedema and displacement of the soft palate may obstruct the airway and produce an acute emergency. The term "floating maxilla" is descriptively applied to this type of fracture.

Le Fort II fracture

This fracture involves the bones constituting the central segment of the face. Classically the fracture line runs from the nasal bones downwards and outwards crossing the

FACIOMAXILLARY INJURIES

infra-orbital margin without as a rule involving the main orbital floor. The line of separation runs through the lateral wall of the antrum below the zygomatic process at a higher level than in Guerin's type of fracture. The typical displacement is backwards and downwards with tilting of the whole complex and gagging on the posterior teeth.

Le Fort III fracture

Here the line of fracture starts at the fronto-nasal suture, frequently involving the cribriform plate. It passes laterally through the ethmoid and lacrimal bones across the orbital floor and to the infra-orbital fissure. The zygomas are detached from the frontal and temporal bones at the respective sutures and the whole complex is displaced along the line of the force applied, downwards and backwards as in the Le Fort II group.

In the Le Fort II and III groups the risks of involvement of other important structures make these injuries major problems in treatment and management. When the initial oedema has subsided the elongation and flattening of the face gives rise to the description "dish face".

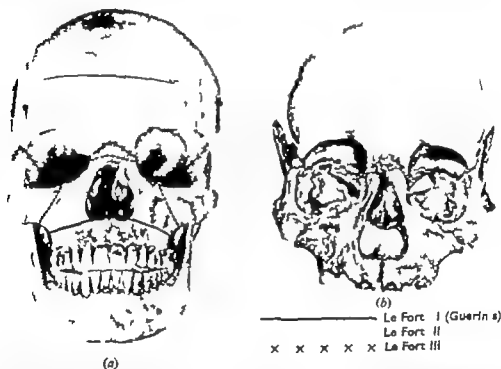


FIG. 74 — Fractures of the middle third of the face. (a) skull showing lines of Le Fort I, Le Fort II, and Le Fort III fractures and (b) reconstituted middle third showing open sutures.

COMPLICATIONS

Certain immediate complications may demand urgent treatment.

Obstruction of the airway

Bleeding may obstruct the nasal passages whilst the oral airway may be reduced by posterior displacement of the mandible or maxilla, with the attached tongue or

TREATMENT

soft palate Foreign bodies such as teeth fillings and dentures may enter the pharynx or larynx In the unconscious patient the dangers are great but the risk can be greatly reduced if the patient is transported and nursed with the face dependent until some temporary control of the fragments and tongue is obtained and all foreign bodies recovered from the mouth The passage of a Magill's tube through the nose is occasionally advised but it tends to become obstructed by blood clot Its insertion may aggravate haemorrhage and increase the risk of infection.

Haemorrhage

When the mandible is fractured large haematomas may arise from rupture of inferior dental or lingual vessels but spontaneous arrest usually occurs In the maxillary complex laceration of soft tissue is seldom gross and persistent bleeding is uncommon Control of bony fragments even of a temporary nature is helpful if bleeding persists.

Interference with coagulated blood in the nose may only aggravate bleeding, but gentle cleansing of the oral cavity can be carried out with the aid of swabs soaked in saline or other bland solution Suction should be available for the aspiration of blood and mucus from the pharynx

Shock

Shock is not a severe or prominent feature of mandibular fractures as such and is very variable in fractures of the middle third of the face Severe shock should suggest the possibility of other injuries

Cerebrospinal fluid rhinorrhoea

Cerebrospinal fluid rhinorrhoea is a frequent occurrence in fractures of the middle third of the face owing to the involvement of the anterior fossa through the cribriform plate Recognition may be difficult in the presence of bleeding or obstruction of the anterior nares by blood clots if doubt exists it is safer to institute prophylactic treatment

Fixation of the fracture will help both to control a leak and to avoid the pumping action of the mobile maxilla which encourages the spread of infection Urgent consideration should be given to definitive treatment taking into consideration the general condition of the patient

The risk of meningitis is always present (see Chapter 17) and there is some divergence of views upon the advisability of dural repair The opinion of a neurosurgeon should be sought when clinical evidence suggests a leak of cerebrospinal fluid.

TREATMENT

The treatment of major jaw injuries requires team work of a high order Much of the final treatment necessitates preliminary technical procedures for the fabrication of splints

The dental surgeon should be consulted as soon as possible so that suitable splints and appliances can be ready for use when definitive treatment is possible

FACIOMAXILLARY INJURIES

The reduction and fixation of fractures of the jaws is greatly facilitated by the presence of teeth. In the great majority of cases the aim will be simply to restore the relationship between the teeth, natural or artificial, of the two jaws. If this can be done the bony fragments will be correctly aligned.

Intermaxillary fixation is the basis of most treatment. On occasions it is supplemented by fixation to a plaster head cap. Occasionally direct fixation of the fragments across the fracture line is advisable.

The majority of fractures in civilian practice are of the simple type, but where comminution exists fragments attached to periosteum, even if small, should be retained. When there is any considerable loss of bone every endeavour should be made to retain the main fragments in their correct relationship to the opposing jaw and associated soft tissues. Sometimes it may be necessary to obtain temporary coverage of the bony skeleton by suturing skin to oral mucosa pending further plastic surgery rather than to aim at a premature reconstruction which might result in permanent deformity.

It is important to reiterate the need for care in diagnosing underlying injuries to the facial skeleton before soft tissue repair is carried out. Consideration may then be given in consultation with others in the team to the possibilities of simultaneous repair of the hard and soft tissues.

Fragments of teeth, fillings and dentures, in addition to the more usual foreign bodies, may be found in the soft tissues and it should be remembered that modern denture materials are not radio-opaque.

If the fracture line is compound into the mouth some degree of infection is inevitable but, in most cases, it can be controlled by immobilization, attention to oral hygiene and the use of a suitable antibiotic. Foreign bodies constitute a potential focus of infection and special consideration must be given to teeth involved in the line of fracture. The dictum that "all teeth in the fracture line should be removed" is generally sound, but antibiotic prophylaxis against infection has made it possible to save a number of these teeth, either temporarily to assist in the immobilization of a fragment or even permanently if aesthetics or function make it desirable. Further supervision and treatment of the teeth will then be necessary.

A few fractures with minimal displacement may be treated without fixation, a semi-solid or fluid diet is then prescribed. This may be especially important in the ill patient, the elderly patient, or the edentulous patient, in whom the inconvenience of treatment with complicated appliances may outweigh the advantages. Minor discrepancies in the edentulous patient can be accepted and adjusted in dentures.

Fractured portions of alveolus or unilateral fractures of the maxillary complex may be immobilized to the remaining sound portion, thus dispensing with the need for intermaxillary fixation, except possibly in the early stages.

Some delay in reduction and fixation may be justified when the general condition of the patient is poor or when gross oedema is present. Disimpaction of middle third fractures however becomes extremely difficult after a few days and may be impossible without direct surgical intervention after two or three weeks.

Impacted fractures of the middle third of the face

To reduce impacted fractures of the middle third two pairs of Walsham's nasal forceps or other similar forceps specially designed for the purpose are commonly

used. The flat blades are inserted along the nasal floor one on each side and the other blades protected by rubber are inserted into the mouth taking care to avoid direct contact with teeth or splints. The surgeon stands behind the patient. The maxilla is grasped with the forceps and is rocked from back to front and from side to side in an endeavour to disimpact it. In fractures of the Le Fort II type disimpaction and reduction of displacement may be difficult until the inwardly collapsed zygomatic arches have been elevated.

Lion forceps one blade in the labial sulcus and one protected in the palate may also be used. Considerable force may be required and care and experience are necessary to avoid adding to existing damage. For this reason more gradual traction by intermaxillary elastics or in difficult cases by the use of weights on a Balkan beam is worth consideration.

Open reduction may be done through the buccal sulci when comminution of the antral walls or infra-orbital floors has taken place. The reduced fragments are retained in position by packing the reconstituted antrum with gauze impregnated with Whitehead's varnish for about 10 days. Fragments of comminuted bone should be retained. If the main fragment of the maxilla is loose packing of the antral cavity may displace it downwards and prevent its reduction so that preliminary fixation of this component may be necessary.

METHODS OF FIXATION

Much of the planning of treatment depends on a knowledge of technical considerations and on co-operation between the surgeon, dental surgeon and technicians. Only the common methods of fixation can be outlined here and much must be left to the experience and dexterity of the individual operator.

Jaws containing natural teeth

Interdental wiring

Two gauges of soft stainless steel wire or brass wire (0.75 and 0.55 mm) should be available.

Direct wiring—A length of wire is passed round individual teeth or groups of teeth and twisted to fit closely around their necks. Immobilization is obtained by twisting together the ends of the various loops in the upper and lower jaws. A clove hitch may be used for single teeth to give improved fixation (Fig. 75a).

Eyelet wiring—The two ends of eyelets previously prepared are passed between two teeth: one end is brought back through the space distal and the other through the space proximal to the space used for insertion. After passing one free end through the eyelet the free ends are twisted together so drawing the wire tightly around the necks of the teeth, and the eyelet into the interproximal space.

Two or more pairs of these wires placed opposite each other in the upper and lower jaws and wired together after reduction is complete, provide very satisfactory fixation (Fig. 75b and c).

This method, the one in most common use, has the great advantage over direct wiring that separate wires are used for the intermaxillary fixation. These are easily replaced should they break and the intermaxillary fixation can be released for examination and replaced if necessary without damage to the eyelet wires.

Arch wiring—This method can be used when there are insufficient sound teeth to permit the simpler wiring methods and no facilities for the construction of splints.

FACIOMAXILLARY INJURIES

A flattened piece of soft German silver wire is bent as accurately as possible to fit the buccal aspect of the arch of teeth to be immobilized. It is wired in position with 0.35-mm gauge stainless steel wire, one turn being passed round the tooth and the second turn around the arch wire. It provides rigid immobilization and has some advantages over other methods but is tedious to fit. Intermaxillary fixation is provided by wires or elastics threaded over the arch wire and attached to the appropriate arch or eyelet wire in the opposite jaw (Fig. 75d)

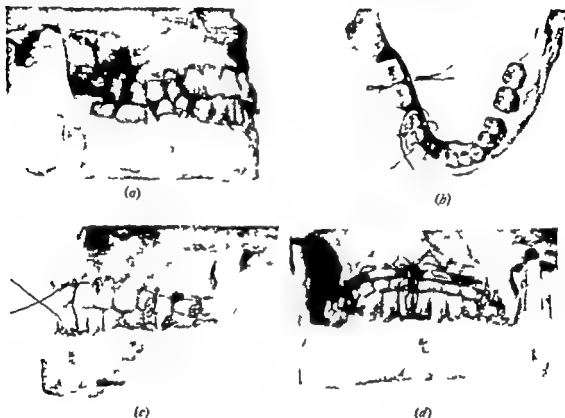


FIG 75—Methods of wiring (a) direct wiring (b) and (c) stages of eyelet wiring and (d) arch wiring.

Silver cap splints

Silver cap splints are used when the number and distribution of teeth is such that the use of interdental wires is not feasible or when treatment is likely to be prolonged and the reduction and fixation difficult.

The technicalities of the construction of these splints are not described except to mention that separate impressions of all groups of teeth in the various fragments of the fractured jaws are taken. Separate splints are then cast in silver alloy covering all teeth up to the gingival margins. To these splints may be added various attachments to facilitate intra-oral and extra-oral fixation. The possibilities of this method are only limited by the number, condition and position of teeth remaining. The technique requires the assistance of a trained dental technician and a properly equipped laboratory. Such splints can usually be constructed in less than 12 hours.

When completed, the splints are fixed to the teeth with dental cement. After 2 or 3 hours the cement becomes hard enough for the splints to be subjected to considerable strain without becoming dislodged.

After reduction of the fracture intermaxillary fixation is completed with wire or elastic bands between the hooks on the splints. Connecting bars precisely made for each case may later be substituted for the intermaxillary wires or may be used to provide rigid fixation across the fracture line. Attachments can be made for these splints to enable them to be articulated by rods and universal joints (Fig. 76) or by wires passed through the cheek to plaster head-caps or to pins screwed into the maxilla or mandible. In this latter connexion some form of insulation must be provided to avoid electrolytic reactions which cause necrosis around the pins if they are left in circuit with the intra-oral splints.

FIG 76.—Skull showing plaster head-cap related by means of rods and universal joints to silver cap splints on the teeth, and a pin in the malar bone



Edentulous Jaws

The absence of teeth removes the most convenient fixed points for the immobilization of the jaws. Methods have accordingly been devised for the fixation of dentures or their equivalent to the jaws and they give a satisfactory method of splintage and intermaxillary fixation. The Gunning splint is the basis of most of the treatment of the edentulous patient. It is therefore important at an early stage to establish that a patient does in fact possess artificial teeth. If he does and they are not with him on arrival at the hospital, steps should be taken to recover them, even if broken as a matter of urgency. If dentures are not available splints may be made to models of the jaws any defects in alignment will be reproduced in the impression and it is necessary at the time of operation to adjust any errors. Black gutta percha is usually preferred for this purpose although other impression compounds may be used.

Gunning splints when completed provide room for feeding at the front and can have extra reduction hooks or locking devices provided as with the silver cap splints. They are fixed to the mandible or maxilla by means of circumferential wires in the lower jaw and transalveolar wires in the upper jaw (Fig. 77). A combination of silver cap splints and Gunning splints may be required where edentulous fragments exist or where one jaw only is edentulous.

FACIOMAXILLARY INJURIES

Skeletal fixation

During and since World War II greater use has been made of the surgical as against the purely dental approach to the immobilization of jaw fractures and two methods are now in common use (a) open reduction and direct wiring of fractured fragments and (b) external pin fixation.

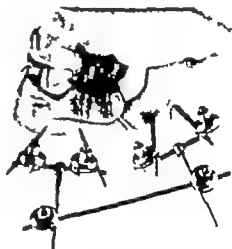
Open reduction and direct wiring—The fracture is exposed intra-orally along the alveolar border extra-orally along the lower border of the mandible, or for the maxillary complex along the infra-orbital margin or the zygomatic arch. The dental drill is convenient for drilling holes through which wires are threaded.

It is frequently desirable to combine this method with some form of intra-oral support, at least in the early stages until some consolidation has taken place.



FIG 77—Model showing Gunning splints wired to the maxilla and the mandible and articulated with black gutta percha.

FIG 78—Model to demonstrate the external pin method of fixation



External pin fixation—This method, originally described by Roger Anderson for the fixation of bones of the leg, was applied to the treatment of jaw injuries during World War II with considerable success. It still has an application in civilian practice when other methods of fixation are impracticable. Originally pairs of smooth stainless steel pins 2 mm. thick were inserted into each fragment and connected by universal joints and bars, each pair of pins in turn being joined by a connecting bar (Fig. 78). The tendency today is to use thicker threaded pins (3 mm. in diameter).

This method of fixation has certain disadvantages. It is cumbersome from the patient's point of view; it is prone to infection and loosening of the pins, and usually requires support from other more conventional intra-oral appliances. It is used when other

FACIOMAXILLARY INJURIES

patients possess a full complement of teeth and there is always space for a semi-solid diet which can be taken through a feeding cup or straw or with the aid of a small pointed spoon. It is never necessary to extract teeth to provide an entry for food. Feeds should be frequent and as varied as possible. Milk products and eggs form the basis of the diet but imagination on the part of the nursing staff and dietician may do much to relieve the boredom of such a regime. Vitamin intake may be augmented and concentrated high protein milk extracts and alcohol to stimulate the appetite are useful additions. Body weight checked twice weekly gives some guidance as to the suitability of the diet. There may be some loss of weight in the first few days until the patient becomes accustomed to the new routine.

BIBLIOGRAPHY

- Fry W. K., and Ward, T. (1956) *The Dental Treatment of Maxillo-Facial Injuries* 2nd ed. Oxford. Blackwell.
Rowe, N. L., and Killey H. C. (1955) *Fractures of the Facial Skeleton* Edinburgh Livingstone.

CHAPTER 20 PERIPHERAL NERVE INJURIES

J. S. HORN

THE MAIN trend in the attitude towards peripheral nerve injuries is from pessimism to qualified optimism. Until World War I nerve injuries were widely regarded as essentially untreatable and attention was focused on stabilization and muscle transfer operations to mitigate their effects. The large number of peripheral nerve injuries sustained during that war necessitated their segregation and special study. It is fortunate that the intellects of Riddoch and Trotter were brought to bear on the problems for even though the inter war years saw a marked decline of interest in peripheral nerve injuries, the foundations were laid for the intensive and well organized research which was one of the best features of the home based medical work during World War II.

The results of treatment of a very large number of nerve injuries at special centres in Britain during World War II were presented in a Medical Research Council report in 1954. They provide the basis for the optimism which should characterize a modern attitude to peripheral nerve injuries.

CLINICAL DIAGNOSIS

Within a few minutes of interruption of nerve conductivity no matter from what cause, the denervated skin becomes flushed and dry. This is the most reliable and most easily observed indication of nerve injury yet it is frequently overlooked in the patient's position. The patient is anxious not to mislead the doctor but at the same time finds it difficult to understand the purpose of the examination. If this is to establish the presence or absence of anaesthesia, repeated and patient explanation will be necessary before the patient understands that he is not being asked whether a stimulus feels normal or abnormal but whether he is aware of it at all. He should map out with his finger the area of altered sensation and then watch the doctor use the hair or pin to test its quality. Checking the information thus obtained by repeating the process with the patient's eyes closed is the final step—not as is so often the case the first step.

Unless precautions are taken to ensure that all extraneous stimuli are eliminated it is easy to conclude that sensation remains when in fact anaesthesia is complete. If the pulps of the fingers are tested with the hand resting on a table the pressure of the pin will be transmitted to normally innervated skin on the dorsum of the hand and the patient will truthfully say that he feels the prick. Similarly if pressure of the pin causes movement at the interphalangeal joints the patient will be aware of deep sensation.

20—A.S.M.

PERIPHERAL NERVE INJURIES

The solution is to hold the part in a firm circumferential grip which avoids all extraneous movement and which allows the pin to test only what it is supposed to test. Even then it requires a conscious effort on the part of the surgeon to avoid tightening his grip synchronously with application of the pin.

With small children it is easier. One dispenses with explanations and relies instead on subterfuge and an absence of adult face freezing stoicism. The concealed pin pricks cautiously outwards from the anhydrotic area while the face is watched for an unequivocal answer.

In testing muscles the golden rule is to see or feel the muscle belly contract rather than to observe the movement it is supposed to have produced, for trick and supplementary movements are common. Trick movements are all passive and are produced either by unparalysed antagonists stretching paralysed muscles, by gravity or by rebound owing to normal elasticity. The last named can be particularly deceptive. Even though the extensor pollicis longus muscle is completely paralysed, extension at the interphalangeal joints of the thumb can be simulated by a momentary and imperceptible flexion movement of the joint, stretching the dorsal structures and producing an elastic rebound. Supplementary movements are produced actively by innervated muscles taking over the function of paralysed ones. Thus the brachioradialis muscle can flex the elbow in the presence of paralysis of the elbow flexors and the supraspinatus muscle alone may be able to abduct the shoulder. Supplementary movements, however, always lack power and they can usually be eliminated by suitable posturing.

Paralysis of deeply placed muscles must perforce be detected by their effect on function and anomalies of innervation are sufficiently common to make diagnosis difficult and uncertain. It is therefore useful to remember that the ability to flex the terminal joints of the index and little fingers nearly always depends on intact median and ulnar nerves respectively.

Of all the tests of movement in the hand, that of Froment is most useful. With both hands the patient pinches a slim book between thumb and index finger. If the short thumb flexor muscle is paralysed or weak, the interphalangeal joint of the thumb will surely flex, for there is no muscle capable of preventing the proximal phalanx from being pushed backwards into extension at the metacarpophalangeal joint by the force of the pinch.

To unravel the complexities resulting from anomalous and dual innervation and from partial denervation selective procaine blocks of the nerve trunks (Higbet, 1942) are sometimes indispensable. Five per cent procaine solution should be used: the injection must be accurately placed and a full 10 minutes must elapse between injection and testing.

NERVE REGENERATION

The process of regeneration is a two-fold one of re-establishing peripheral connexions and functional maturation of the conducting apparatus.

Within 2-3 days of a nerve crush the zone of destruction is crossed by very fine threads of axoplasm which arise by the splitting of a single axon into numerous fish-like processes. In the initial phases this axoplasmic outflow is at the expense of depletion of the proximal axon, but later it is continued by synthesis of new material. Schwann cells in both proximal and distal stumps proliferate very rapidly

NERVE REGENERATION

and by migration cross the zone of destruction. The products of myelin and axon degeneration are phagocytosed by mesodermal elements and Schwann cells throughout the length of the distal segment enlarge both in length and in thickness so that their protoplasm fills the lumen of the Schwann tubes.

If the severed ends of the nerves are in contact a small proportion of the brush like axonal outgrowths find their way into the Schwann tubes of the distal segment. The remainder after a futile search involving the evolution of bizarre forms atrophy and disappear. Two or more axons may enter a single Schwann tube but only one survives.

If on the other hand the ends of the nerves are separated characteristic changes occur in each. In the distal end a phase of Schwann cell proliferation is followed by fibroblastic invasion from the surrounding non neural tissue and this converts the end of the nerve into a somewhat shrunken adherent scarred structure to which the name glioma has been somewhat incongruously applied. At the proximal end intense and prolonged activity results from the outgrowth of axoplasmic processes unrestrained by Schwann tubes and this eventually results in the formation of a false neuroma. The cut ends of the nerve fasciculi seem to terminate in a fan like fashion but beyond this the rapidly growing brush-like axoplasmic threads thrust out in all directions forming an irregular maze as though searching in vain for a guiding tube. They angle sharply turn back on their tracks and spiral round their proximal course to produce "spirals of Perroncito". Schwann cells also proliferate in an unorganized manner and both neural and glial elements evoke a vigorous fibroblastic response which serves to limit their migration and to enclose them in a fibrous bulb. This bulb—the false neuroma—develops a more or less well defined capsule although it always remains to some extent adherent to the surrounding tissue. The maze of fine axons within the bulb remain unmyelinated, except near the proximal cut end where some are enclosed in thin sheaths and many of them disintegrate under the pressure of the enveloping scar tissue.

There is a continuing tendency for axonal growth into the neuroma and although with the passage of time the proportion of scar tissue to axons increases it is possible that false neuromas continue to grow albeit at a progressively slower rate where gross continuity has not been lost. The rate of axonal growth varies according to the nature of the lesion and the condition of the distal segment. Lytton and Murray (1954) have shown that hyperplasia of Schwann cells in the distal segment facilitates axonal downgrowth and that cortisone administration by depressing the Schwann cell hyperplasia which normally accompanies Wallerian degeneration slows down the rate of re-innervation of the degenerated nerve. There is no evidence that the level of the lesion has any significant effect. In rabbits an average rate of down growth after section and suture is 3.5 mm a day and after nerve crush, 4.4 mm a day (Young 1942). There are no reliable figures for man and it is probable that, although average rates are considerably slower than those in rabbits there are great variations.

The fibres first laid down are very small in diameter and have a correspondingly slow conduction rate. The process of maturation involves a progressive increase in fibre diameter and although this may continue for many years it is rarely complete. The degree of functional recovery is closely correlated with the restoration of fibre diameter and since the latter is usually defective so is the former.

PERIPHERAL NERVE INJURIES

The nature of the regenerating fibre—its diameter and whether or not it is myelinated—depends on its central connexions and not on the characteristics of the Schwann tube. Conversely its peripheral connexions and the character of new end-organs formed in response to the arrival of regenerating fibres depend on the type of tissue being re-innervated and not on the central connexions of the axon. It is likely that there is selective resorption of fibres establishing useless peripheral connexions.

It is widely assumed that a divided nerve cannot regenerate unless it is sutured. Nevertheless an injury which divides both deep and superficial flexor tendons in a finger frequently also divides one or both digital nerves and one has been repeatedly struck by the fact that the patients when seen at a later date rarely have anaesthesia of the finger. In one case in which an operation note made at the time of injury stated that all six digital nerves in three fingers had been divided, very full spontaneous recovery of sensation occurred. Young (1942) has shown in the dog that proliferation of Schwann cells from the distal stump of a nerve can create new segments of nerve up to 3 cm. in length, the direction of outgrowth of Schwann cells being largely determined by pre-existent tissue planes.

Spontaneous restoration of continuity of divided digital nerves in the fingers is doubtless facilitated by the well defined tissue planes in which these nerves lie, but there is no reason to doubt that the same thing does not occur elsewhere. Indeed it would be surprising if it were not so for even the most meticulous nerve suture produces only a relatively close approximation of nerve bundles, and it has been claimed that a gap of up to 3 mm. between the nerve ends after nerve suture has no adverse effect on the result (Clifton 1949).

Sunderland (1947) has pointed out that a large proportion of nerves found in continuity at operation show some spontaneous recovery even though the appearances strongly suggest that the nerve had been completely or partly divided at the time of injury.

It is reasonable to assume therefore, that under suitable conditions divided nerves can regenerate without suture although the quality of recovery is likely to be defective.

OPERATIVE TREATMENT

Controversy centres chiefly around the questions of immediate or delayed suture after traumatic nerve section, the place of neurolysis, and the problem of the non-conducting nerve in continuity.

Primary versus secondary nerve suture

Seddon (1949) basing his opinion on the difficulty of predicting the extent of the intraneural scar which inevitably develops on the danger of sepsis following suture of lacerations, and on the contradiction between the need for mobilization of the nerve on the one hand and the non-desirability of exposing uninjured tissue planes to the risk of infection on the other, strongly advocated delayed suture. His views were naturally influenced by the fact that he was largely concerned with war injuries where the risk of sepsis was high and intraneural damage extensive.

OPERATIVE TREATMENT

For civilian nerve injuries it seems reasonable to perform delayed suture when ever the risks of failure of primary suture are high either because of the nature of the wound or the level of the injury. A high primary nerve suture is not intrinsically more likely to fail than a low one but evidence of its failure will not be forthcoming for a longer period. This delay will seriously jeopardize the prospects of a second operation and for this reason even favourable types of high nerve section are best repaired after an interval of 2-3 weeks from wound healing.

On the other hand clean cuts of the median or ulnar nerve above the wrist can be sutured primarily with an excellent chance of success. Usually only a minimum of mobilization is necessary and with careful technique aided by antibiotic protection sepsis should not occur. Successful primary suture not only avoids a second operation but also enhances the quality of the result by minimizing anastomotic confusion due to rotation of the nerve ends for it is usually easy to see which way the nerves should lie at the primary operation. If owing to intraneural scarring recovery is not manifest within 2-3 months delay of this order does not significantly impair the results of a second operation.

Primary suture is indicated for digital nerves because of the technical difficulties of delayed suture. Unfortunately many lesions of digital nerves are due to blunt destructive injuries, such as those caused by high speed circular saws and primary suture is not usually possible. However if the nerve ends are excised and laid in their correct tissue plane with the finger in moderate flexion recovery is surprisingly good and often follows within 2-3 months. If recovery is inadequate continuity can best be restored by using free grafts to bridge the gap.

Plasma clot 'suture'

Since the pioneer work of Young and Medawar (1940) many workers have investigated the possibilities of utilizing the adhesive qualities of clotted plasma to secure apposition of the nerve ends. The theoretical advantages are that it is less damaging to the nerve since it is unnecessary to pass needles and suture material through the nerve sheath that it leaves no foreign material at the suture site and therefore reduces the amount of post-operative scarring, and that if properly applied it can ensure more uniform apposition of the nerve ends.

Basically all methods of plasma clot suture depend on the conversion of fibrinogen into fibrin either by the addition of thrombin or in the case of oxalated plasma, by the addition of calcium. As a result a fibrin clot forms within a period which varies from a few seconds to several minutes and if during this time the nerve ends are held in apposition and completely immersed in the developing clot more or less firm adhesion results. To localize the clot to the suture line it is sometimes necessary to create a temporary artificial 'lake' by the use of wax or Stent moulds.

The main disadvantages of plasma clot suture are that it is often difficult and time consuming to secure neat apposition and that the tensile strength of the resultant suture is considerably less than that which can be obtained by ordinary suture. Not only is the union less strong immediately after it is completed but a natural accession of strength is delayed. The thickness of the clot around the apposition line prevents an early fibroblastic reaction of the surrounding tissues from contributing effectively to the maintenance of contact. A further drawback is that retraction of the nerve sheath, which may be quite marked is not prevented by plasma clot

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suture as it is by conventional suturing and this theoretically increases the risk of regenerating nerve fibres finding their way out of the nerve sheath

Fine stainless steel wire tantalum and the newer synthetic fibres combine considerable tensile strength with a high degree of inertness and therefore conventional suturing with these materials is preferable whenever there is more than minimal tension on the nerve ends—and this is the case with the majority of delayed nerve sutures.

Plasma clot is of great value for the repair of small nerves like digital nerves, where it is technically very difficult to insert a sufficient number of conventional sutures without injuring the nerve ends. It may also facilitate cable and inlay grafting procedures.

Neurolysis

Although the therapeutic value of neurolysis has been questioned and even categorically denied it can on occasion produce a complete reversal of the clinical condition. This happy result does not depend on liberating the nerve from strangulation by scar tissue or callus, for when such gross pathology is found the outcome of the operation is usually disappointing. It must have a subtler basis, for the operation is most successful when the naked eye pathology is minimal.

Exploration for persistent ulnar nerve paralysis following trauma behind the elbow may show no pathological changes in the nerve, but it is frequently followed by an immediate return of sensation to the ring and little fingers and by a somewhat slower return of motor function in the hand.

Similarly re-exploration of a sutured median nerve which has failed to recover within the expected time has been followed by immediate recovery even though no abnormality was seen at operation. On one occasion the same effect was produced by percutaneous procaine infiltration around the suture line.

Such cases are difficult to explain but they have occurred too frequently to be dismissed as coincidences. It may be that freeing the nerve relieves spasm in the vasa nervorum and so improves the blood supply in the vicinity of the injury. It has been shown that moderate ischaemia of a nerve can block conduction without causing Wallerian degeneration and that the vasa nervorum are under vasomotor control (Adams 1942).

The non-conducting nerve in continuity

There is a growing tendency with which the author is in agreement, to explore the majority of closed nerve injuries which fail to manifest signs of recovery within 3-4 months irrespective of their level and of their expected date of recovery. The rationale for such a policy is the relative rarity of true axonotmesis and the adverse effects of delaying nerve suture particularly for high lesions beyond 6-9 months. In consequence one increasingly sees lesions in continuity which necessitate an immediate decision as to whether they should be excised and sutured or whether spontaneous recovery can occur if they are left. Among the factors on which such a decision can be made the consistency of the lesion is the most definite. A hard lesion implies impenetrable intraneural scar tissue which will prevent recovery unless excised.

Trial incision of the lesion to arrive at an estimate of the number of bundles

traversing it is a useful and surprisingly safe method if properly performed. If the lesion is stretched over a finger and incised transversely with feather strokes of a razor the natural plane of cleavage between intact bundle and enveloping scar tissue opens up when it is reached and is easily visible. In general if 50 per cent of bundles are intact resection should not be performed.

Direct electrical stimulation above the lesion may result in a flicker of contraction in distally innervated muscles, in which case spontaneous recovery is probable.

If joint stiffness and contractures have already occurred excision and suture of a high lesion of a predominantly motor nerve is probably contra-indicated whatever the local pathology for by the time axons grow down the condition is likely to be irreversible. In such a case one should consider whether a muscle transfer might not restore better function than nerve suture. On the other hand restoration of continuity of the median nerve is nearly always worth attempting, for there is no other way to return sensation to the hand and even very imperfect sensibility is better than none at all.

A point worth bearing in mind is that the result depends to a considerable extent on the technical excellence of the suture. However this crucial step in the operation coming at a time when the surgeon has been strained and fatigued by a long and difficult dissection is something of an anticlimax and is apt to be differently performed. It is, therefore, often better to hand over this part of the operation to an assistant—a course of action which may also pay dividends in the final steps of a finger flexor tendon grafting operation.

NERVE GRAFTING

Autogenous nerve grafting

Autogenous nerve grafting has now been established as a reliable procedure which when competently carried out for correct indications can give results comparable to nerve suture under the same conditions. The most frequently available type of graft is the cable graft made up of strands of cutaneous nerves. It is better not to use cutaneous nerves innervating skin bordering on the denervated area, even though this may be technically more convenient for the added disability of extending the anaesthetic area may be significant. Most of the available cutaneous nerves are 2-3 mm. in diameter and since they have a tendency to shrink they should be cut 15 per cent longer than the gap to be bridged.

Inlay grafting

Inlay grafting with strands of cutaneous nerves is particularly useful for partial lesions of nerves where, after excision of a lateral neuroma, a useful proportion of bundles is found in continuity even though there may be a complete physiological block. A plane of cleavage is established between interrupted and intact bundles and the former are resected until both cut surfaces are free from fibrous tissue. Undegenerated bundles will then put out from the proximal end while the degenerated bundles in the distal segment will appear as semi-translucent pinkish dots. It is particularly important to resect the distal segment down to unequivocally healthy tissue. Inlay grafting is a far more satisfactory procedure than folding the intact portion of the nerve into a loop so as to make possible suture of the divided bundles.

icle grafting

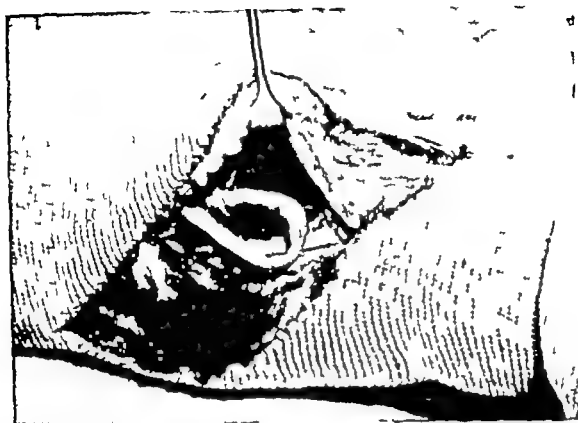
edicle grafting (Strange 1947) is a most useful advance. It was originally to make good defects in the median nerve when both median and ulnar d sustained appreciable loss of tissue, by utilizing the proximal portion ar nerve as a graft in a two-stage operation. The author has used it for electrical burns of the forearm, for industrial injuries, and for the nerve lesions of Volkmann's contracture. The longest graft measured d every case which has been followed up has had a reasonably good return n (Fig. 79).

1 of Volkmann's contracture

t operation for correction of Volkmann's contracture is often a radical of necrotic muscle and scar tissue with the primary object of correcting (Seddon 1956). During this operation, the median and ulnar nerves are and if it is clear that irreversible changes have occurred in both, the final d be the establishment of a median-ulnar anastomosis above the level hological changes and division of the ulnar nerve high enough in the i to ensure that the length of ulnar nerve graft available is amply sufficient the gap in the median nerve. Subsequent operations may include a full-flap skin-graft to provide gliding tissue and tendon transfers grafts or to restore finger flexion. During all these procedures the ulnar nerve it left undisturbed *in situ*. Median nerve axons are continually penetrating length and the less the nerve is subjected to operative trauma and the emans in a more or less normal environment, the faster this penetration lace. Provided the skin and tendon operations are completed reasonably as operation of bringing down the ulnar nerve graft and anastomosing istal segment of the median nerve should be performed as the final step onstructive procedure. A biopsy specimen from the terminal end of the e graft taken during this last operation may show that the vanguard have already penetrated its full length, in which case the time when the of sensory recovery in the hand may be expected to appear can con : predicted. Success in the operative treatment of Volkmann's contrac res radical surgery as early as possible without wasting time on such procedures as elastic stretching, tendon lengthening, muscle slides, and

TRACTION INJURIES OF THE PERONEAL NERVE

ion peroneal nerve has the unique anatomical characteristic of pursuing course between two bony points: the ischium and the head of the fibula. eason it is particularly susceptible to traction injuries. Moreover, in view ight course, it is very difficult to gain length for nerve suture, and this is the main reason why the results of suture of this nerve are notoriously ngston (1947) reported that among more than 300 major nerve lesions resection and suture, only 10 cases failed because of disruption at the : but that 9 of these 10 cases involved the peroneal nerve. Whitcomb orted that 70 per cent of all suture line disruptions involve the peroneal



(a)



(b)

FIG. 79 — Pedicle nerve grafting. (a) Anastomosis between the median and ulnar nerves. (b) The graft in place. The distal suture line is visible at the lower extremity of the wound.

It is therefore advisable to gain length for suture of the peroneal nerve by resection of the head and neck of the fibula rather than by flexing the knee otherwise the tension thrown on the nerve during the period of knee straightening will either disrupt the suture line or cause a diffuse traction lesion in the nerve.

Two kinds of closed traction lesions of the peroneal nerve are well known and have been reported in the literature. They are due to fracture of the pelvis with displacement and to adduction type dislocation of the knee with rupture of the fibular collateral ligament.

The author has encountered two additional mechanisms, one proven and the other not yet proven which have not previously been reported.

Retrograde pull on the nerve by muscle action

Case report

A middle-aged woman slipped in the street and sustained an adduction sprain of the ankle. On examination, she stated that the entire lower limb below the knee was numb and paralyzed. Perception of pin prick was lost in the distribution of the peroneal nerve and reduced elsewhere. She could not voluntarily contract any muscles below the knee. There was swelling below the lateral malleolus and radiological examination showed that the talus could be tilted in the ankle mortise. There was tenderness over the head of the fibula.

Six hours later there was full recovery of function in the posterior tibial nerve but the peroneal nerve remained paralyzed.

Two days later peroneal nerve paralysis remained complete and ecchymosis had appeared over the upper part of the peroneal muscles. The nerve was explored and it was found that the peroneal muscles had been partly avulsed from their fibular origin and had dragged the peroneal nerve distally. Some muscular branches of the nerve had been torn and there was extravasation of blood beneath the nerve sheath extending as far proximally as the bifurcation of the sciatic nerve into its two main branches.

In the course of a 2 year follow-up the patient made only a partial recovery of function.

This then was a case of traction injury of the peroneal nerve owing to retrograde pull on it by the peroneal muscles during forced inversion of the foot. The initial total paralysis of the leg was probably due to a transient transmitted lesion of the posterior tibial nerve.

Similar but slighter cases have also been seen most of them resulting in degenerative paralysis of the extensor hallucis longus muscle only.

Prolonged non-violent stretching of the nerve

The second as yet unproven, mechanism of traction injury of the peroneal nerve is prolonged non-violent stretching of the nerve by flexion of the hip with the knee extended.

It has been known for many years that patients confined to bed for long periods were liable to develop paralytic foot drop. This was regarded as a reflection on the quality of nursing care and the solution was thought to be the provision of foot cradles to keep the weight of bedclothes off the feet and blocks to prevent plantar flexion of the ankles. However even the most assiduous nursing was not always successful in preventing this complication.

SPLINTAGE

It is also well known that patients on traction are liable to develop foot drop. It is suggested that in both types of case paralysis is due to prolonged gentle stretching of the peroneal nerve produced either by sitting up in bed with the knee straight or by excessive elevation of the straight leg.

The evidence for this point of view is as follows: (a) Straight leg raising is known to throw tension on the peroneal nerve and its diagnostic value in cases of prolapsed intervertebral disc derives from this. (b) The foot drop is a true paralytic phenomenon with sensory loss in the appropriate area. Sometimes the biceps femoris muscle is also paralysed, proving that the cause cannot be pressure of a Thomas sling on the peroneal nerve at the neck of the fibula. (c) Since it was first suspected 6 or 7 years ago that this may be the cause of foot drop, the author in treating fractures of the femur with the knee in full extension has avoided suspending the Thomas splint above an angle of 20 degrees with the bed, and no further cases of foot drop have occurred. (d) A patient with a supracondylar fracture of the femur was treated by traction with the knee in moderate flexion. Four weeks later when the knee was brought into full extension, paralytic foot drop promptly developed. The angle between the Thomas splint and the bed was found to be 55 degrees.

SPLINTAGE

The role of splintage in the treatment of peripheral nerve injuries was for many years grossly exaggerated as a result of a widespread belief that unless paralysed muscles were uninterruptedly held in a state of relaxation they could not recover. This edict must have been propounded with weight and authority for it has not only found its way into innumerable textbooks, but has also achieved the distinction of becoming one of the indispensable facts that medical students and nurses were expected to learn. Happily the edict is not true.

Were it a fact that intermittent stretching of a paralysed muscle has a deleterious and lasting effect on its chance of recovery, an insoluble contradiction would present itself. If the paralysed muscles were uninterruptedly splinted in relaxation to give them a chance to recover stiffness and deformity of the limb would inevitably develop. Conversely, if passive movements were employed to preserve mobility this would ensure permanent paralysis for passive motion must inevitably stretch the paralysed muscles.

The cult of relaxing paralysed muscles lingers on, but consideration of two common lesions should help its demise.

Sciatic nerve injuries result in paralysis of both plantar flexors and dorsiflexors of the ankle. As the patient lies in bed with his foot dangling helplessly into equinus deformity, it is self-evidently correct to splint the foot at right angles to the leg. This relaxes the tibialis anterior muscle, but at the expense of stretching the calf muscles. Yet it is the latter which have the better chance of recovery.

Dislocation of the shoulder is often complicated by paralysis of the deltoid muscle. With the arm bandaged to the chest, the paralysed deltoid muscle is stretched to the maximum. An abduction frame would partly relax it, but at the risk of redislocation—an exorbitant price which no surgeon should incur on his patient's behalf. Happily in the vast majority of cases the deltoid muscle recovers and a normal shoulder results.

Prevention of deformity

The main role of splints in the treatment of peripheral nerve injuries is to prevent deformity. Fixed deformity following peripheral nerve injury arises from one of two sources, or a combination of both. If the paralysed muscle is opposed by a bulky and powerful antagonist the latter is very likely to become progressively shortened and to produce fixed deformity.

If the unparalysed antagonists are relatively less bulky if they normally come into action only intermittently for the performance of specific movements and particularly if the limb is persistently used by the patient as normally as possible, the tendency to muscle shortening is much less. Radial nerve paralysis resulting in wrist drop is alleged to result in fixed shortening of the wrist flexors. Such shortening develops early and to a marked degree if the wrist is neither splinted nor used. If it is splinted and exercised for purely prophylactic purposes, muscle shortening is retarded and reduced in severity but if the hand is used for hard manual work from soon after the onset of paralysis then irrespective of whether or not a splint is used shortening is unlikely to develop. The author recently saw a Chinese peasant with a complete radial nerve paralysis of 9-years duration. Throughout this time he had continued to till the land and to reap its produce. There was not the slightest trace of shortening of the wrist flexors. Every time he had thrust his spade into the earth or steadied his carrying pole on his shoulder his wrist had been carried into full passive extension. His complaint was of weakness of the grasp for when he clenched the fist the wrist went into flexion and the efficiency of the finger flexors was markedly impaired.

Joint contracture

The other and probably more frequent cause of fixed deformity after peripheral nerve injury is joint contracture. Brachial plexus lesions may be rapidly followed by stiffness of the fingers owing to peri-articular thickening consequent upon immobility combined with oedema. Joint contractures in isolated median or ulnar nerve injuries develop as a result both of postural deformity and vasomotor abnormality in the denervated area, for they appear much more rapidly than can be explained by postural deformity alone.

Ulnar nerve paralysis

In the claw hand of ulnar nerve paralysis the metacarpophalangeal joints especially of the fourth and fifth fingers are pulled into hyperextension by the long extensor muscles unopposed by the paralysed interosseus muscles. With the metacarpophalangeal joints extended, the long flexor tendons are passively stretched and pull the interphalangeal joints into flexion a process which is facilitated by loss of the extending action of the interosseus muscles on the interphalangeal joints. After a time which may be as short as 2-3 months, the volar capsule of the interphalangeal joints shortens and produces fixed limitation of extension at the interphalangeal joints.

It is frequently recommended that to prevent this a splint should be worn to keep the metacarpophalangeal joints flexed. However such splints are cumbersome and may prevent resumption of work. Moreover since the recovery of intrinsic muscle function following ulnar nerve injury is notoriously defective in

quality it is not possible to assure the patient that he will be able to dispense with the splint within a reasonable time. An additional reason for avoiding the routine use of ulnar nerve splints is that in the author's experience functionally significant deformity by no means regularly develops without their use. Bowden (1954) reported that 75 per cent of patients with ulnar nerve injuries who did not use splints developed contractures of the fourth and fifth fingers but she does not indicate the severity of the contracture or the effect it had on function. Usually there is only a trifling flexion contracture in the proximal interphalangeal joint of the little finger. Passive flexion at the metacarpophalangeal joints is not usually limited for the extensor tendons can be relaxed by the merest dorsiflexion of the wrist. Even though the loss of dexterity resulting from paralysis may be very great joint contractures may not add significantly to it. The reason why fixed deformity is often so slight in ulnar nerve lesions is doubtless that in a large proportion of cases the interossei muscles receive a dual innervation from both median and ulnar nerves.

However in a minority of cases presumably those in which the interossei muscles are exclusively ulnar innervated severe deformity and disability develop early. Active flexion at the metacarpophalangeal joints is impossible and the grasp function of the hand is practically lost. Correction of the hyperextension deformity at the metacarpophalangeal joints is urgently required in such cases. A knuckle-duster splint can achieve this as a temporary measure but since the functional recovery following nerve suture is not likely to be satisfactory early operative intervention to restore muscle balance may be indicated. The simple procedure described by Zancolli (1957) of shortening the volar capsule of the metacarpophalangeal joints may provide a solution for these difficult cases. Should this operation fail it is still possible to undertake the more complex procedure devised by Bunnell (1944) in which the terminal bifurcations of the sublimis tendons are inserted into the dorsal expansions.

To summarize it may be stated that the only fixed deformity in most cases of ulnar nerve paralysis is flexion contracture of the proximal interphalangeal joint that most cases of ulnar nerve paralysis do not need splintage that the minority of cases who do require splintage require it for gross impairment of the grasp function of the hand and that in these cases early operation to prevent hyperextension deformity of the metacarpophalangeal joints is probably indicated.

Median nerve paralysis

The motor consequences of median nerve injury on the function of the hand are very variable but if the power of opposition is lost the effect on function is serious and a splint should be worn pending either muscle recovery or a tendon transfer operation. If a splint is not used fixed deformity nearly always develops and negates the benefits either of eventual re-innervation or of a tendon transfer. Again, the cause of fixed deformity lies both in joint changes and in muscle contractures. The thumb metacarpal bone is drawn into an adducted externally rotated, and extended position and shortening of the capsule of the carpometacarpal joint and of the adductor pollicis muscle prevent passive opposition. When this has happened tendon transfers to restore opposition are doomed to failure unless they are preceded by restoration of free mobility to the thumb metacarpal

PERIPHERAL NERVE INJURIES

bone, and this necessitates capsulectomy of the carpometacarpal joint in addition to tenotomy of the adductor pollicis muscle

Probably a higher proportion of opponens muscle paralyses than of interosseus muscle paralyses require splinting. Unfortunately a really satisfactory opponens muscle splint has not yet been designed for it is very difficult to maintain opposition without straining the metacarpophalangeal joint of the thumb

RESULTS OF NERVE SUTURE

The results of nerve suture in 1,441 carefully documented and followed up patients treated at five Medical Research Council nerve injuries centres during World War II have been analysed by Zachary (1954). These were mostly war injuries and therefore sepsis, delay in treatment, loss of nerve tissue, and extensive muscle damage were common. A similar series of civilian nerve injuries treated with equal skill would certainly give better results. Nevertheless, the overall picture that emerges is very encouraging and should do much to stimulate a more active and hopeful attitude to the treatment of peripheral nerve injuries.

Some of the salient points in this analysis are as follows.

Ulnar nerve

Following low suture under favourable conditions 78.5 per cent made a useful degree of recovery in the intrinsic muscles of the hand and 16 per cent recovered independent lateral movements of the fingers. The overall figure for high and low sutures performed both early and late was that nearly 50 per cent had a useful degree both of motor and sensory recovery.

Median nerve

Of low early sutures 68 per cent made useful sensory recovery and 53 per cent of all cases did so. Of high sutures 90 per cent recovered useful finger flexion but only 32 per cent of all cases recovered useful opposition of the thumb.

Radial nerve

Of radial nerve cases 89 per cent recovered useful power in the wrist extensors and 61 per cent also recovered power in the finger and thumb extensors. Fine control with complete co-ordination of extension of fingers and thumb was achieved in 36 per cent of cases.

Medial popliteal (posterior tibial) nerve

Of medial popliteal nerve cases 56 per cent recovered powerful action of the calf muscles against resistance. Toe flexion rarely returned and only 27 per cent of patients had a return of tactile sensibility in the sole of the foot although 80.5 per cent were able to perceive pin prick.

Lateral popliteal (peroneal) nerve

Of lateral popliteal nerve cases 36 per cent recovered the power to dorsiflex the ankle against gravity but only 13 per cent could do so against moderate resistance.

Sciatic nerve

The interesting and important fact emerged that out of 107 cases of sciatic nerve suture 45 per cent recovered good function. Good function was defined as no foot ulcers, no more than minimal pain, and ability to walk more than 1 mile and to do work involving a fair amount of standing and walking.

Twenty nine patients made no neurological recovery at all, but of these 27.5 per cent had good function as defined above. Conversely a high degree of neurological recovery did not necessarily imply a return of good function. The conclusion can therefore be drawn that although in general the better the neurological recovery the better the functional recovery, this is not always so and an irrecoverable lesion of the sciatic nerve should never be regarded by itself as an indication for amputation. The main safeguards against amputation are meticulous foot care and splintage to prevent deformity.

REFERENCES

- Adams, W. E. (1947) *J. Anat., Lond.* 76, 323.
 Bowden, Ruth E. M. (1954) *Rep. med. Res. Coun. Lond.*, No 282, p. 326. London: H.M. Stationery Office.
 Bunnell, S. (1944) *Surgery of the Hand*. Philadelphia: Lippincott.
 Clifton, E. E. (1949) *Surgeons*, 26, 756.
 Highet, W. B. (1947) *J. Neurol. Psychiat.*, 9, 101.
 Livingston, W. K. (1947) *J. Neurosurg.*, 4, 16.
 Lytton, B., and Murray, J. G. (1954) *J. Physiol.*, 126, 627.
 Seddon, H. J. (1949) *Brit. J. Surg., War Surg. Suppl.* 2, 375.
 — (1956) *J. Bone Jt. Surg.*, 38B, 152.
 Speransky, A. D. (1944) *A Basis for the Theory of Medicine*. New York: International Publishers.
 Strange, F. G. St. C. (1947) Preliminary communication. *Brit. J. Surg.* 34, 423.
 Sunderland, S. (1947) *Brit. J. Surg.*, 35, 36.
 Whitcomb, B. B. (1946) *J. Neurosurg.*, 3, 399-406.
 Young, J. Z. (1942) *Physiol. Rev.*, 22, 319.
 — and Medawar, P. B. (1940) *Lancet*, 2, 126.
 Zachary, R. B. (1954) "Results of Nerve Suture." *Rep. med. Res. Coun. Lond.*, No 282, Chap. 8. London: H.M. Stationery Office.
 Zancolli, E. A. (1957) *J. Bone Jt. Surg.*, 39A, 1076.

CHAPTER 21

REHABILITATION OF THE INJURED

R. BARRIE BROOKES

INTRODUCTION

MUCH has been written about rehabilitation many definitions have been suggested and lengthy explanations given of its many implications. It is a word which has been so continuously before the public that its practical aims are sometimes in danger of being taken for granted. In its broadest sense it summons up a picture of the work associated with world wide problems of social upheaval following World War II. In its narrower meaning it provides the bridge on which the injured worker must pass from hospital bed to factory bench. It is in this latter sense that the part played by the rehabilitation "get fit" team is now considered.

With an increase in responsibility and equipment and the expansion of her training curriculum the masseuse graduated to the status of physiotherapist and the massage department became a new-styled physiotherapy department. By the same token there might be a case now for thinking of physiotherapy departments as rehabilitation units. There is a growing tendency towards the revaluation of many forms of treatment taught under the generic heading of physiotherapy. Dead wood is being rejected and indications more accurately defined.

At the same time it is recognized that occupational therapy which was once mainly concerned with mental illness, can play a decisive part in assisting the recovery of physical function. Certain handicrafts were introduced and modified for this purpose, but after early enthusiasm this has been much criticized. It was often associated with an unrealistic attitude which involved treating the condition rather than the patient.

The active procedures of physiotherapy and occupational therapy have been increasingly geared to the working, domestic and social requirements of the individual. There is no longer a stereotyped method of handling, for example a united fracture of the tibia. The object of therapy is in fact to rehabilitate John Smith to return to his work as a maintenance engineer after he has broken his leg. The possible tasks of treatment are illustrated by a fireman with many years of service to his credit. While at work he fell from a height and sustained severe bilateral calcaneal fractures. Within twelve months he was back at full work after a rehabilitation programme which started a few days after injury with non weight bearing exercises in bed and concluded with running, skipping, jumping and climbing with and without weights.

For a man working at a sedentary job reasonable ambulation is all that may be necessary to get him back to work, but if a fireman cannot return to full duty it means the loss of pension and other rights to which he is entitled by his long service. For the same degree of recovery rehabilitation must be considered successful in the clerk but a failure in the fireman.

INTRODUCTION

The end result must be one that is acceptable to the patient. A restricted range of movement in which he has confidence and which will allow him to earn his living is better than a wider range without confidence in its use.

Therapists must never allow a state of inertia to develop whilst awaiting instructions. They should be encouraged to use ingenuity and skill to plan a progressive programme of graduated activity within a broad prescription which might simply be "progressive weight bearing".

Teamwork

Successful rehabilitation can only come from the closest integration between physiotherapist, occupational therapist and remedial gymnast as part of a medical team. With a broader conception of the contribution each can make to the individual's needs, each of the therapies become less exclusive in its contribution, resulting in a certain amount of overlap. The distinctive skills and specialized methods all aim to develop the patient's personal independence by retraining that may first involve the activities of daily living and self care but which leads eventually to a final resettlement in a constructive life even though this may fall short of the pre-accident state.

It is an advantage in interpreting the vocational needs of the injured worker in any industrial area to have a working knowledge of the various trades, and particularly of the tradesmen. A therapist who is able to show that he knows something of the working conditions to which the patient will return has the advantage of getting on intimate terms with him so that his resources can more easily be developed. The patient will gain confidence when he knows where he is going.

Most patients associate severe injury with the possibility of being unable to return to their former work. The fear is that alternative work will mean reduced earnings. Even if he can return to his old job, the patient may be anxious about his ability to hold it down. A therapist who has had a look at local industries can go a long way to dispel much of the psychological overlay and thereby remove one of the barriers to progress. There is no single factor more important than this ability of the therapist to get close to the patient. Academic qualification is of no avail without it, and is the reason why rehabilitation can succeed in little more than four walls, yet fail in the best equipped department if the right attitudes are missing.

Perhaps in the past some players on the rehabilitation team have been inclined to keep to their set positions too rigidly and to insist that the other players do the same. Teamwork never will reach its full target even when the special needs of the patient have been established, unless each member is prepared to do that little bit extra to bring it about.

Now that the modern trend of treatment is to make the patient ambulant as early as possible, it is never easy to get the more helpless ones sitting out of bed, particularly when they are overweight. This calls for teamwork. The problem should never be left entirely to the nurse to solve as part of her nursing duties any more than it is regarded as the exclusive province of the physiotherapist or remedial gymnast. Anything other than a combined effort from all who are working on the ward will militate against the progress of the patient.

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Co-operation with Industry

An important aspect of teamwork is its extension into industry and its acceptance there by every working man and woman.

A machine operator sustained a fracture of the wrist which was treated in plaster. He was advised to return to work in order to make full use of his fingers and shoulder. On his return to hospital several weeks later for the removal of plaster his fingers were swollen, stiff and painful and he could not raise his arm without pain in the shoulder. The foreman had taken the trouble to put the man on a machine the controls of which had been altered so that it could be worked with one hand, to enable him to rest the injured one in a sling all day!

This misdirected sympathy added several months disability to a man who should have resumed his normal productive capacity within a short time of union of the fracture.

Attitude of relatives

The public at large have also a part to play and can make or mar attempts at rehabilitation. 'Killing with kindness' can be a reality when those who have the care of a relative with a disability following injury stifle attempts at independence with the mistaken idea that they are being helpful. The man who has lost an arm will never feel that he has made the grade until he is allowed to put on his own coat without someone continually rushing to help him.

A youth was discharged from hospital several months after a severe head injury. After prolonged unconsciousness and confusion he was eventually able to walk unaided, climb stairs and care for himself generally. He also undertook the normal hospital schooling. On discharge he was left with some spasticity of the left arm and leg and slowness of mental response. When his mother arrived to take him home she dressed him completely. She had arranged a bed for him downstairs and was obviously aiming to do everything for him.

The mother had not realized her key position in her child's rehabilitation. His complete independence was to depend upon her encouragement and patience together with the right amount of parental discipline.

PHYSIOTHERAPIST AND OCCUPATIONAL THERAPIST

In any formulation of general principles it would be invidious to distinguish too closely between the roles of the physiotherapist, occupational therapist and remedial gymnast. Each aims at encouraging the activity of the patient and directing it into useful channels.

The inco-ordinated patient will still be taught to carry out Frenkel's exercises by the physiotherapist and a suitable craft by the occupational therapist but at this point each worker will lose her identity as she becomes concerned in ensuring that the patient can also carry out the every-day activities of caring for himself. It has been the failure to carry treatment to this stage which has provoked criticism in the past. Functional activities must be fully understood by all therapists and the basic principles merit inclusion in the training curriculum at the expense of some of the never used Swedish remedial exercises.

Physiotherapy

Few observers would deny that there are many patients suffering from the after-effects of injury who do not need physiotherapy. In these days of over crowded departments the careful assessment of each injury is an essential part of their administration so that the available time will be used to do a good and complete job with the more severely disabled patients. Time might otherwise be spent to less effect on a dozen "quick turnovers". One of the most important contra indications for physiotherapy still remains the absence of any need for it.

Group treatment

Physiotherapy for trauma is essentially a matter of correct timing and no department should ever be adding names to waiting lists. As the basis for treatment however is active exercise the development of group and class work has become an integral part of the rehabilitation programme and should make waiting unnecessary. Careful planning must go into the preparation of exercise schemes and once a patient has been individually instructed and understands what is required of him he can usually continue exercises in a class with other patients having similar aims. A traumatic effusion of the knee joint is commonly seen. Once the patient has learned quadriceps muscle contractions his exercises can be supervised in an early class. As the effusion subsides progression will be made to a class which combines the former exercises with knee flexion and then according to individual requirements, will progress to resisted and agility exercises combined with climbing as indicated. A ladder is an essential item of equipment for lower limb rehabilitation.

With up to four or five classes for each anatomical region it is possible to have a smooth progression from an acute condition to final recovery for many patients at any one time provided that the therapist in charge of each class is aware of the limited objectives of that class.

Exercise at home

For many patients one or perhaps two attendances suffice for exercise instruction. When a patient is working and it would be economically difficult for him to attend a few and only a few specific exercises are taught for him to practise regularly throughout the day. When possible it is advisable to have clear instructions printed and handed out to the patient and explained to him individually.

It is essential that the patient is not told simply to use the part but shown the actual range through which the limb should be moved. Every patient with a wrist in plaster for example, is not just told to move his fingers but is also shown how to put his hand behind his neck and in the hollow of his back to prevent the shoulder becoming stiff and painful.

Experience has shown that when a middle aged or elderly person sustains injury to the shoulder the degenerative changes of the rotator cuff consistent with this age group take unkindly to insult, and rupture of the musculotendinous structure is frequent. It is important for exercise to begin within a few days of injury in order to recover function but it is wrong to tell the patient vaguely to keep his shoulder on the move. If the patient is not shown exactly how to carry out the exercises and particularly what movements to avoid his shoulder will more than likely become

stiffer and more painful in the succeeding weeks. He must be weaned from the over use of a sling and the use of heat and other passive therapies when the acute stage has passed. Unless active exercise forms the keystone of treatment, it will be doomed to failure.

Occupational therapy

In recent years occupational therapy has largely left behind its array of weaving and rug-making machines which were adapted to produce any movement specific for a particular injury but took little account of the job to which the patient was returning. The occupational therapist has progressively modified the work to a modern functional approach with the growing provision of more varied occupational aids including the facilities for helping the disabled housewife. Nor are the methods confined to the department but reach out also to the home and business premises of the patient.

It is true that for a large proportion of patients recovery can be harnessed to function at the earliest possible time by return to work, modified if necessary or possible. This will complete the cure as quickly as any formal rehabilitation programme. There is however a hard core of patients recovering from injury for whom occupational therapy is desirable.

There is the patient who cannot yet do his own work and for whom suitable work is not available yet progressive activity is his main need for recovery. There is the reluctant patient whose apprehension amounts to inhibition, and there is the one who requires retraining to become independent again before industrial resettlement can be considered.

There is no doubt that to prepare a man for his earliest return to work the most satisfactory method is for him to handle tools comparable with those of his own work and in an atmosphere of normality. He is more likely to respond to the stimulus of work he understands and finds satisfactory than to using apparatus which is obviously intended to recover lost range in a joint or to strengthen a muscle group but which he finds frustrating on account of its unfamiliarity. More satisfactory results come from using the affected joint or muscle group as a co-ordinating part of the limb as a whole. It is in an attempt to fulfil this requirement that occupational therapy departments now devote much space to the use of whatever engineering tools and practices are possible working with both wood and metal materials.

Aids to daily living

For the patient who has lost some of the skill needed for independent existence, therapists concentrate on the early attainment of complete self care. Facilities are provided to take these patients through the motions of toilet activities, eating and drinking, dressing and undressing. Such patients are put through such day-to-day hand activities as striking a match, handling door knobs, window fasteners, electric switches, telephone dials, taps and everything else which is commonly found in the house and may be appropriate.

Just as the industrial patients are rehabilitated with the tools of their trade so also is the housewife. Many departments have now established a kitchen as part

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of the rehabilitation facilities in which the housewife may be taught to recover her dexterity

It is a comparatively simple matter to provide a gadget to help a patient carry out an essential function which otherwise he could not do. A long handled shoe horn will overcome the disability of a stiff hip and a wooden extension arm from a tap will assist a person with poor hand function.

It would be a complete negation of all these attempts at rehabilitation if a patient discharged home from hospital is unable to carry out the activities which he has been encouraged to adopt. A high bed may make it difficult for him to get in and out by himself. A polished floor on which he has already slipped destroys his confidence to move about freely. Such difficulties can mean an individual becoming bedfast so that rehabilitation has failed in its final objective. It should be possible to ensure that these preventable hazards do not interfere with progress and they can often be overcome by a home visit.

Rehabilitation of patients with limb injuries

The hand and foot serve to show how a purposeful programme of activity can be planned along functional lines to secure the greatest recovery from injury.

The hand

It is to the hand as an organ carrying out the intricacies of commission expression and appreciation that man owes his supremacy in the animal kingdom. Any part which is destroyed cannot be replaced but even when severe mutilation has become an efficient instrument with which a man can once more earn his living (Fig. 80a-e).

The joints of the upper limb have been developed so that by their close coordinated action the hand can be placed flat on any surface of the body with the exception of a small area between the scapulae. This enables it to be used to advantage in all activities of the body.

When the implications of severe trauma become obvious to a patient the mind receives a scar possibly more disabling than the one on the body (Lan. 1950). The unduly large share of the hand in cortical representation can make the problems of replacement more intractable than with any other part of the body. The patient's first reaction is often to keep the hand out of sight in a pocket or glove believing it to be cosmetically repulsive and functionally useless. Encouragement is not always easy and progress cannot begin until the individual accepts the hand as the one he must live with. But as introspection is overcome by the skill of the therapist a definite plan of rehabilitation is started.

As an organ of commission, the hand must be able to perform five principal functions, namely (1) a clamp for holding down (Fig. 80b) (2) a vice for gripping (Fig. 80c) (3) a pincer for fine holding (Fig. 80d) (4) a hook for carrying (Fig. 80e) and (5) a sensitive antenna for feeling, upon which all the foregoing functions depend.

The restoration of these functions must become the final aim of treatment. Experience has shown that when the hand is capable of carrying out these functions,



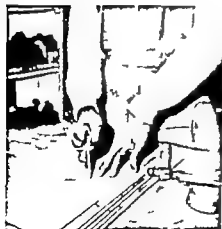
(a)



(b)



(c)



(d)



(e)

Fro 80 —(a) A hand injury (b) The clamp (c) The vice. (d) The pincer
(e) The hook.

regardless of the degree of injury few obstacles will remain to prevent the worker returning to his original work.

The question of right or left handedness is an important one. The dominant hand should be retained as the master whenever the main functions can be recovered with intensive retraining. The patient will usually find this an easier task than to change the respective roles of the dominant and subservient limbs. A right handed person will find it simpler to write again even with a mid forearm prosthesis than to start from the beginning with the left hand. With a pencil fixed to a leather cuff fastened around the stump in such a case writing practice can start even before the removal of sutures, effective movement coming entirely from the shoulder joint.

The foot

Subtalar joint mobility —The foot has been developed by the body as a means of propulsion and balance. Injuries impairing its function will in consequence deprive the individual to a varying extent of the ability to walk, run, jump, climb and balance. Disability arises in the main from restriction of movement in the subtalar joint, which is the joint of balance, and weakness of the posterior tibial group of muscles. Many patients with this disability have had a fracture-dislocation of the ankle or a fracture of the calcaneus. When patients with either of these injuries reach the weight bearing phase they are often unable to balance on the injured foot or rise on the toes. From this clear aim of treatment have emerged Exercises must emphasize the development of balance, standing and raising the body weight on the toes. During the period before weight bearing is allowed but when the joints are no longer immobilized in plaster, the essential exercises concentrate on mobility of the subtalar joint and strengthening the posterior tibial muscles. While adequate movement in the ankle joint is equally important for good walking, this usually recovers rapidly once weight bearing begins.

An ankle which has been injured and immobilized is usually stiff and may require intensive assistance to mobilize the subtalar joint. On the other hand when active movements are permitted immediately for a calcaneal fracture the maintenance of joint range is at this stage the most important aim of the therapist. Ultimate function depends to a large extent on the mobility of this joint.

Plantar flexion —It is equally important that resisted plantar flexion exercises are begun as soon as possible. All injuries are associated with some reflex inhibition of muscle tone and in this neighbourhood this may lead to atrophy of the posterior tibial muscles. Exercises must be carried out to the point of maximum load consistent with the injury and the comfort of the patient. Muscle will hypertrophy only in direct proportion to the amount of resistance it works against and free exercises are useless for this end when the muscle group concerned is capable of harder work.

Attempts have been made in calcaneal fractures to relieve the lesion from the adverse effects of weight bearing but to preserve a degree of function by getting the patient to walk on the toes of the affected foot. It is quite impossible in most cases, however, for this to succeed owing to the immediate tonal loss of the muscles of plantar flexion.

In the final phase of rehabilitation all the exercises are concerned with balance and toe-standing, and include climbing exercises. There seems little purpose in teaching a patient to use his foot like a hand, picking up objects with the toes,

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as is done to strengthen the intrinsic muscles in cases of flat feet, when these important muscles are necessarily used powerfully in the foregoing exercises.

Swelling—Swelling is a common sequel to injuries of the lower leg. Massage, elevation, pressure bandaging, faradism under pressure, sinusoidal baths and exercises can all be used to advantage in certain circumstances but when swelling is more than transient there is nothing as effective as the circumferential pressure exerted by an efficient elastic stocking which should extend from the web of the toes to above the upper margin of the oedema. It is advisable to wear the stocking until it has worn out, by which time it will probably be no longer required.

AMBULATION

The teaching of a patient to walk again following a lower leg injury has many aspects, some of which have not received the attention they warrant. The complete scheme involves early ambulation out of bed, perhaps within a day of operation,

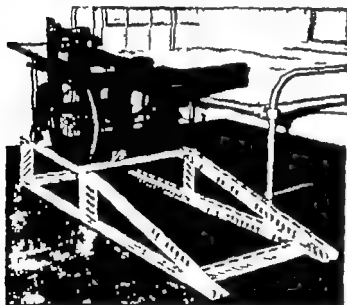


FIG 81—Modified wheel chair and bedside ramp

followed shortly by instruction in the use of crutches. Thereafter the usual pattern of progress follows with partial weight bearing between crutches, then on two sticks graduating to one stick, and finally to full weight bearing without support. It is often stated that the patient should have two sticks or none but in actual practice the giving up of one stick forms a mark of progress to the patient. Held in the opposite hand the remaining stick forms a satisfactory support without imbalance and it does mean that more weight is put on the leg than might otherwise be the case with two sticks. So many patients walk badly with no sticks and well with one stick, that the procedure is amply justified.

Even when a formal rehabilitation programme is not required, the issue of crutches, sticks and plaster overboots should be done from the rehabilitation department. The patient who requires support should be shown how to use it. Often in the transition to weight bearing close supervision is necessary untoward reactions must be looked for and the degree of weight bearing controlled accordingly.

AMBULATION

Getting the patient up

More remains to be done to facilitate getting heavy and awkward patients out of bed. Those who do the lifting often themselves develop backstrain. Hoists are now available and when much heavy lifting is required they should be part of the ward equipment. It is possible to improvise from steel tubing and clamps a frame which will wheel over the bed and from which a winding mechanism can be suspended for such lifting.

An alternative and efficient method has been developed with a modified wheel chair and ramp (fig. 81). The ramp at the side of the bed enables the chair to be brought up to bed height. The back support folds flat and the leg support is raised. With the arm rest removed it is a relatively simple matter to move the patient onto this chair. It is then reconstituted and wheeled down the ramp with the patient upright.

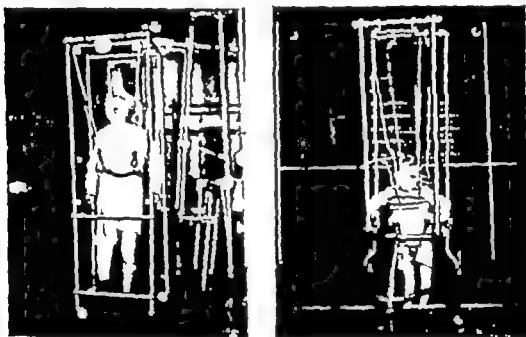


FIG. 82.—Adjustable walking apparatus adapted to prevent full weight bearing during walking.

Walking apparatus

Several weeks of ambulation time can often be usefully saved in bilateral lower limb injuries by the use of a walking apparatus in which the patient is supported by a sling around the pelvis and between the legs. When the full body weight cannot be taken by the leg and four point walking with crutches is not practical the sling ropes can be adjusted to allow no more body weight on either leg than is necessary for the foot to make contact with the ground for propulsion. The ropes can be adjusted to allow increased weight bearing as indicated.

This apparatus can also be constructed from tubing and clamps and is completely adjustable (Fig. 82).

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Special problems

There are two particular hazards which the more seriously injured individual encounters after discharge from hospital but which can be largely overcome by instruction. Many patients lack the initial confidence to step on and off a bus and so hesitate to use public transport. The provision in the department of a bus entrance on which a patient can practise largely overcomes this.

Difficulty which may be experienced in mounting a pavement can also be overcome by using a platform at the correct height in the department. This handicap may be found when both knees are stiff from injuries involving both legs.

REFERENCE

Lane, R. W (1950) *Physiotherapy* 36, 11

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